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TELEROENTGENOGRAPHY OF THE HEART IN BERIBERI

T. K. RAMAN, M.D., D.T.M.

From the Department of Medicine, Andhra Medical College and King George Hospital, Vizagapatam

Historical

Roentgenological observations of the heart in beriberi were made by Muira (1909), Ido and Watanabe (1914), Reinhard (1916) and Alsmeer and Wenckebach (1929). Definite enlargement of the right side of the heart was observed by all in the majority of patients (quoted by Keefer). Keefer (1930) from a study of eight cases found definite increase in the size of the heart in all of them. The enlargement was observed mainly in the right side of the heart. Prominent pulmonary conus and the shadow of the superior vena cava were observed in addition. After treatment there was decrease in the size of the heart with disappearance of the shadow of the pulmonary conus and superior vena cava. In eight cases that did not show any signs of cardiac insufficiency the increase in size of the heart was observed only in 2 cases.

Chopra *et al* (1937) from a teleroentgenographic study of the heart in sixty cases of epidemic dropsy found enlargement of the right auricle and the left ventricle in 70 per cent of the cases. In addition, prominent pulmonary conus was seen in a few cases.

Material for Investigation

The present series include 15 cases. All of them were clinically and therapeutically proved cases of beriberi. These were definitely not cases of epidemic dropsy. Almost all of them had œdema, tachycardia and peripheral neuritis and all of them were cured with vitamin B_1. Epidemic dropsy cases are never cured so easily. Peripheral neuritis is also a rare condition in epidemic dropsy. The photographs were taken at two meters distance in the standing or in the sitting posture when the patient was too ill to stand up. The method of Vacquez and Bordet was employed in taking the measurements. The four points A, B, C, and D are first marked out and measurements taken (*vide* Diagram I).

Case 1—The patient gave a history of beriberi of two years' duration. The present attack started one year ago with œdema, tachycardia and peripheral neuritis. The heart was enlarged and systolic murmurs were heard in the mitral and pulmonary areas. Gallop rhythm was present—rate 120 p.m. B. P. 130/60 m.m. of Hg. Calf muscles were tender and deep reflexes were lost. He was put on vitamin B_1 injections for two days (2 mgm). On the third day the pulse rate came down to 60 and the blood pressure to 120/80. (Figures 1, 2, 3). Three pictures were taken on 26-9-40, 30-9-40 and 7-10-40. The first picture showed enlargement of both the right and the left side. Subsequent teleroentgenography showed reduction in the transverse diameter, left more than right, reduction in the total length (longitudinal diameter), reduction in the size of all chambers of heart in the following order: (*a*) right ventricle, (*b*) right auricle (*c*) left ventricle (*d*) left auricle and reduction in the bisector (thickness of the left ventricular muscle). Reduction in the second week was much more than that observed during the first week.

Case 2—The patient gave a history of illness of 15 days' duration and showed œdema, tachycardia and peripheral neuritis. He had only 5 injections of vitamin B_1 of 5 mgms. each. Three pictures were taken in this case also on 22-9-41, 3-10-41 and 13-10-41 respectively. Here also the subsequent picture showed reduction in the transverse diameter, left more than the right, slight reduction in the total length with slight relative increase in the ventricular length and insignificant increase in the size of right auricle and right ventricle. There was first a reduction in the auricle and then an increase, in the ventricle, there was first an increase and then a reduction. There was reduction in the size of the left auricle and no change was observed in the left ventricle. Thickness of the ventricular wall was reduced.

(A) The point of junction of the vascular and cardiac shadows on the right side. (B) The point of junction of the cardiac and diaphragmatic shadows on the right side. (C) The junction of the left auricle or pulmonary conus with the left (ventricular) border. In cases where there is difficulty in locating this point, it is decided by fluoroscopy. (D) The apex or the junction of the left border of the heart and the diaphragm. 1. Right median—the distance between the middle line and the extreme right border of the heart (*a*). 2. Left median—the distance between the middle line and the extreme left border of the heart (*b*). 3. Transverse diameter—the sum of the right and left median. 4. Total length—the distance between the apex of the heart and the point of junction of the right auricle and the superior vena cava (A.D.). This line is divided into two by the middle line and the distance above the division is called auricular (x) and the distance below as the ventricular length (y). 5. A-B represents the right auricular length, B-D the right ventricular, C-D the left ventricular, and the perpendicular distance from C to the middle line, the left auricular length. 6. Right oblique—the perpendicular distance from B to A-D. 7. Left oblique—the perpendicular distance from C to A-D. 8. Bisector—the perpendicular distance from the point of maximum convexity of the left border to C-D. This figure represents the thickness of the left ventricular wall. 9. Aortic breadth—the maximum breadth of the aorta usually taken at the level of the 3rd intervertebral disc. 10. Transverse diameter of the chest—the inner diameter is taken. 11. The ratio of the heart diameter with the total diameter of the chest.

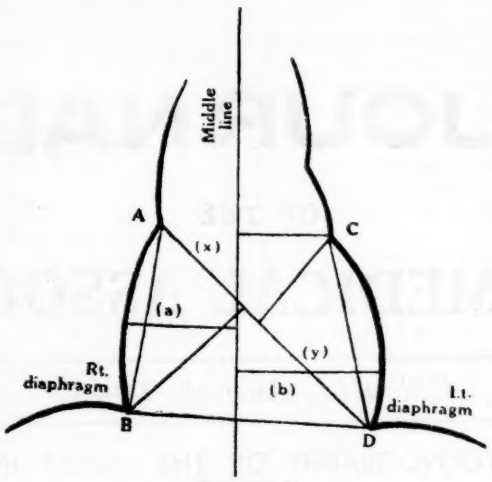

DIAGRAM I.

Case 3—This was a chronic case of 4 years' duration. At the time of admission he showed œdema, tachycardia and peripheral neuritis. B. P. 110/70. He had only 3 injections of vitamin B_1 (5 mgms). Only two pictures were taken in this case, the second one 9 days after the first; the latter showed reduction in the total transverse diameter, reduction in the total length, with well marked reduction in the ventricular length, reduction in the right side of the heart both the auricle and the ventricle and reduction in the size of the left side both the auricle and ventricle. There was definite reduction in the thickness of the ventricular muscle. In addition he showed an aortic knob and a prominent superior vena cava. After treatment the shadow of the superior vena cava disappeared but the aortic knob persisted.

Case 4—The patient gave a history of one month's duration, and showed œdema, tachycardia and peripheral neuritis. He had only two injections of vitamin B_1 (5 mgms). Here also two pictures were taken at an interval of about one week. The second one showed reduction in the total transverse diameter, slight increase in the total length (this was manifested in the auricular length), reduction in the size of all the chambers of the heart and reduction in the thickness of the left ventricular muscle. Superior vena cava was visualised and the aortic knob was prominent.

Case 5—This was the first attack of 3 days' duration. He showed œdema, tachycardia and peripheral neuritis. B. P. 118/68. He had only two injections of vitamin B_1. The peculiarity in this case was that the heart in the first picture apparently looked normal and reduction after treatment showed that normal size was represented by the second picture. There was reduction in the transverse diameter, reduction in the total length, reduction in the size of both sides, right and left, and reduction in the thickness of the ventricular muscle. (*cf.* Figs. 7 and 8).

Case 6—He showed œdema, tachycardia, peripheral neuritis and systolic murmur in the pulmonary area. B. P. 116/50. He had only one injection of vitamin B_1 (5 mgms). Two pictures were taken at an interval of 6 days. Comparison of the pictures (Figs. 5 and 6) showed reduction in the transverse diameter, reduction in the total length with slight increase in the auricular length, reduction in the size of the heart, right as well as the left. There was no change in the thickness of the ventricular muscle.

Case 7—The patient was seen twice and showed on both occasions œdema, tachycardia and peripheral neuritis. He had no injections of vitamin B_1 but was treated only with a liberal diet. The second picture was taken after three months. It looked as if the size was the same as before or there was a slight increase in the size of the heart. When actual measurements were taken the total internal diameter of the chest was more than that in the first picture, and when the figures were corrected after the original picture, reduction in the transverse diameter, reduction in the total length, reduction in the size of the right and left side and reduction in the thickness of the ventricular muscle were observed.

Case 8—This was the first attack and the patient showed œdema, tachycardia and peripheral neuritis. He was treated only with a liberal diet. The pictures taken at an interval of 6 days showed reduction in the transverse diameter, reduction in the total length, and reduction in the size of both the ventricles. There was a slight increase in the size of both the auricles and reduction in the thickness of the ventricular muscle.

Case 9—He gave a history of 1½ months' duration and showed œdema, tachycardia and peripheral neuritis. He had no injections of vitamin B_1 but was treated with only liberal diet. The two pictures taken showed reduction in the transverse diameter, reduction in the total length with slight increase of the auricular length, reduction in the size of the right auricle, left auricle, left ventricle and reduction in the thickness of the ventricular muscle. Prominent pulmonary conus was seen in both the pictures and reduction in the size of the conus was seen in the second picture.

TABLE SHOWING DETAILED MEASUREMENTS OF THE HEART OF CASES REPORTED

Case Nos. Name, Age & Sex.	Dates of skiagraphy	Right median	Left median	Transverse diameter	Total Length	Auricular length	Ventricular length	Right auricle	Right ventricle	Left auricle	Left ventricle	Right oblique	Left oblique	Bisector	Aortic breadth	Total diam. of chest	Ratio of heart to total diam.	Remarks.
Case 1. P. R. 20. M.	26-9-40.	5.7	7.8	13.5	14.5	4.7	9.8	8.5	14.5	5.3	8.1	6.7	4.6	1.1	5.0	22.8	0.58	
	30-9-40.	5.0	7.3	12.3	13.5	4.2	9.3	8.0	11.8	5.0	7.5	7.0	3.8	0.9	5.0	do	0.53	
	1st Reduction	0.7	0.5	1.2	1.0	0.5	0.5	0.5	2.7	0.3	0.6	0.3	0.8	0.2	nil			
	7-10-40.	4.0	6.7	10.7	11.8	3.5	8.3	7.5	9.8	4.5	7.0	6.1	3.8	0.9	5.0	22.8	0.46	
	2nd Reduction	1.0	0.6	1.6	1.7	0.7	1.0	0.5	2.0	0.5	0.5	0.9	0.0	0.0	nil			
	Total Reduction	1.7	1.1	2.8	2.7	1.2	1.5	1.0	4.7	0.8	1.1	0.6	0.8	0.2	nil			
Case 2. N. P. 22. M.	22-9-41.	4.6	8.8	13.4	14	3	11	7.7	12.1	5.0	9.0	6.6	4.0	1.4	5.0	25.7	0.52	
	3-10-41.	4.7	8.4	13.1	13.5	2.7	10.8	7.0	12.7	4.3	9.0	6.5	3.6	1.35	5.0	do	0.51	
	1st Reduction	+0.1	0.4	0.3	0.5	0.3	0.2	0.7	+0.6	0.7	0.0	0.1	0.4	0.05	nil			
	13-10-41.	4.0	8.2	12.2	13.8	2.7	11.1	7.8	11.5	4.8	9.0	6.5	3.9	1.30	5.0	25.7	0.47	
	2nd Reduction	0.7	0.2	0.9	+0.3	0.0	+0.3	+0.8	1.2	+0.5	0.0	0.0	+0.3	0.05	nil			
	Total Reduction	0.6	0.6	1.2	0.2	0.3	+0.1	+0.1	0.6	0.2	0.0	0.1	0.1	0.1	nil			
Case 3. G. R. 30. M.	18-10-41.	4.9	8.6	13.5	14.1	3.2	10.9	8.1	13.0	5.0	8.9	7.2	4.2	1.2	6.2	26.8	0.5	Prominent aortic knob and superior vena cava.
	27-10-41.	4.8	7.2	12.0	13.4	4.0	9.0	8.0	11.8	4.1	8.6	7.0	3.5	0.9	6.2	do	0.44	Prominent aortic knob. The shadow of superior vena cava has disappeared.
	Reduction	0.1	1.4	1.5	0.7	0.8	1.9	0.1	1.2	0.9	0.3	0.2	0.7	0.3	nil	nil		
Case 4. Y. 25. M.	15-11-41.	4.5	8.3	12.8	13.1	3.3	9.8	7.5	12.0	4.7	7.4	6.6	3.4	0.8	5.5	26.0	0.49	Prominent superior vena cava & aortic knob.
	21-11-41.	4.2	7.3	11.5	13.5	3.8	9.7	7.5	10.8	4.3	7.2	6.4	3.2	0.6	5.5	do	0.44	The shadow of superior vena cava has disappeared.
	Reduction	0.3	1.0	1.3	+0.4	0.5	0.1	0.0	1.2	0.4	0.2	0.2	0.2	0.2	nil			
Case 5. G. N. 20. M.	3-11-41.	3.8	8.4	12.2	14.2	4.5	9.7	7.9	11.5	4.9	9.4	6.3	4.0	1.35	5.8	25.8	0.42	The heart in the first instance looked normal but reduction in size after treatment showed that the second picture represented the normal size of the heart.
	11-11-41.	2.7	7.55	10.25	12.2	3.3	8.9	6.1	10.2	4.4	8.8	5.1	4.1	0.95	5.8	do	0.39	
	Reduction	1.1	0.85	1.95	2.0	1.2	0.8	1.8	1.3	0.5	0.6	1.2	0.1	0.40	nil			
Case 6. A. R. 29. M.	23-10-41.	5.9	7.6	13.5	14.5	4.0	10.5	9.0	12.5	4.4	8.5	8.7	3.5	1.0	6.5	27.0	0.49	
	29-10-41.	5.6	6.0	11.6	12.6	4.8	7.8	7.5	10.8	3.5	7.2	6.4	3.6	1.0	6.5	do	0.44	
	Reduction	0.3	1.6	1 9	1.9	+0.8	2.7	1.5	0.9	1.3	2.3	0.1	nil	nil				
Case 7. Ch. A. 23. M.	25-9-41.	5.0	6.2	11.2	13.2	3.0	10.2	8.1	11.4	4.7	8.0	6.9	3.5	1.1	4.7	23.5	0.47	The increase in measurements are not real, but when the figures are corrected for the size of the internal diameter reduction in size was observed.
	26-12-41.	5.0	7.5	12.5	12.5	3.0	9.5	8.3	11.9	3.7	7.5	7.5	2.8	1.1	5.3	27.8		
	Corrected measurement	4.3	6.4	10.7	10.8	2.6	8.2	7.1	10.2	3.2	6.4	6.4	2.4	0.9	4.6			
	Reduction	0.7	+0.2	0.5	2.4	0.4	2.0	1.0	1.2	1.5	1.6	0.5	1.1	0.2	0.1			
Case 8. P. A. 22. M.	10-10-41.	4.2	9.0	13.2	14.0	2.9	11.1	7.1	13.2	4.3	9.0	6.6	3.3	0.8	5.0	24.5	0.53	
	16-10-41.	4.0	8.8	12.8	13.5	2.5	11.0	7.3	12.8	4.4	8.0	6.7	2.5	0.5	5.0	do		
	Reduction	0.2	0.2	0.4	0.5	0.4	0.1	+0.2	0.4	+0.1	1.0	+0.1	0·8	0.3	nil			
Case 9. S. M. 20. M.	11-9-41.	4.0	7.3	11.3	14.0	3.2	10.8	7.3	11.5	3.8	10.8	6.0	4.2	1.3	5.9	24.3	0.46	Prominent pulmonary conus
	19-9-41.	4.2	6.0	10.2	13.5	4.7	8,8	7.1	11.5	3.5	9.5	6.0	3.8	1.0	5.9		0.43	Pulmonary conus present but less marked than before.
	Reduction	0.2	1.3	1.1	0.5	+1.5	2.0	0.2	0.0	0.3	1.3	0.0	0.4	0.3	nil			
Case 10. G. R. 20. M.	2-9-41.	4.1	9.5	13.6	15.5	4.5	11.0	8.3	13.1	4.9	10.5	7.0	4.0	1.4	5.0	24.6	0.50	Prominent pulmonary conus
	9-9-41.	3.2	9.4	12.6	14.5	5.0	9.5	7.2	12.0	4.3	10.1	6.0	3.8	1.4	5.0		0.46	Pulmonary conus present but considerably reduced.
	Reduction	0.9	0.1	1.0	1.0	+0.5	1.5	1.1	1.1	0.6	0.4	1.0	0.2	nil	nil			
Case 11. A. A. 25. M.	25-9-42.	5.1	6.5	11.6	12.5	4.0	8.5	7.9	11.0	3.1	8.8	6.9	3.9	1.2	5.1	24.5	0.47	There was no oedema on both occasions. Tachycardia in the first instance.
	9-11-42.	4.7	6.9	11.6	12.5	3.4	9,1	7.5	11.2	3.2	9.0	6.6	4.0	1.0	5.1		0.47	
	Reduction	0.4	+0.4	0.0	0.0	0.6	+0.6	0.4	+0.2	+0.1	+0.2	0.1	+0.1	0.2	0.0			
Case 12. K. M. R. 18. M.	1-11-41.	4.7	8.0	12.7	14.7	6.0	8.7	9.3	11.0	4.0	10.3	7.0	4.2	1.4	5.2	26.8	0.47	Prominent pulmonary conus
Case 13. G. A. 17. M.	15-10-41.	4.5	7.0	11.5	12.5	4.5	8.0	7.5	10.5	4.0	9.1	6.2	4.1	1.0	4.7	21.5	0.49	Prominent pulmonary conus congestion of both lungs.
Case 14. S. A. 21. M.	20-12-41.	4.7	7.8	12.5	12.6	5.1	7.5	7.5	11.7	3.7	8.3	6.9	3.5	1.2	5.0	28	0.44	
Case 15. M. P. 25. M.	2-12-41.	4.9	7,3	12.2	14.4	5.5	8.9	8.5	11.1	3.7	11.3	6.5	4.7	1.5	6.0	25.5	0.47	Ascending aorta slightly enlarged.

Case 10—This was the first attack and the patient showed œdema, tachycardia, and peripheral neuritis. He had three injections of vitamin B_1 (5 mgms each). The second picture taken after a week (Fig. 4) showed reduction in transverse diameter, reduction in the total length with slight increase in the auricular length, and reduction in the size of all the chambers of the heart. There was no reduction in the thickness of the ventricular muscle. Both the pictures showed well marked pulmonary conus. The second picture showed slight reduction in the size.

Case 11—He gave a history of œdema and showed only tachycardia and advanced peripheral neuritis, at the first and only peripheral neuritis at the second examination. He was treated with a liberal diet and twelve injections of vitamin B_1 (5 mgms). Two pictures were taken at the two visits (interval about 1½ months). The second picture showed reduction in the transverse diameter with relative increase of the right median. There was no reduction in length. The decrease in the auricular length was compensated by increase in the ventricular length. Except the right auricle all the other chambers showed slight increase in size. There was slight reduction in the ventricular thickness.

In each of the last four cases (Nos. 12, 13, 14 and 15) only a single picture was taken. Enlargement of the heart was present in all the cases and two (Nos. 12 and 13) showed prominent pulmonary conus.

The detailed measurements of the heart are shown in the accompanying table.

Discussion

The findings in this series confirm the observations made by others. In acute cases the heart is enlarged both to the right as well as to the left, but when the question arises as to which individual chamber is affected the difference of opinion comes in. In my series of cases, the right heart was enlarged more than the left and the right ventricle more than the right auricle. It is possible for the auricle to be enlarged more than the ventricle in cases where congestive heart failure is present in addition. Thickness of the left ventricular wall is increased. There is no method of measuring the thickness of the right ventricular muscle. The question that arises is whether the measurements described above actually represent the size of the chamber concerned. In case 2, although there was definite reduction in the transverse diameter there was no appreciable decrease in the size of any of the chambers.

The symptoms of beriberi disappeared in the following order: (1) œdema. (2) cardiovascular disturbance. (3) Peripheral neuritis. In Case 19 Figs. 1, 2 and 3

LEGEND FOR FIGURES

Fig. 1. (Case 1) showing enlarged heart both right and left taken on 26-9-40.

Fig. 2. Same as above taken on 30-9-40 showing reduction in size.

Fig. 3. Same as above taken on 7-10-40 showing a still further reduction in the size. Definite reduction in size can be seen when figures 1 and 3 are compard.

Fig. 4. (Case 10) showing enlargement of the heart and prominent pulmonary conus. A second picture taken at the end of a week showed reduction in the size of the heart and reduction of the pulmonary conus. Disappearance of the pulmonary conus showed that the condition is neither congenital nor rheumatic.

Fig. 5. (Case 6) showing enlargement of the heart, both right and left taken on 23-10-41.

Fig. 6. Same as above taken on 29-10-41 showing considerable reduction in size. There is a slightly dilated aorta. This is probably a separate condition not related to beriberi.

Fig. 7. (Case 5) Heart appears practically normal in size taken on 3-11-41.

Fig. 8. Same as above showing definite reduction in size, taken on 11-11-41. The normal size of the heart of this patient is represented by this picture.

showed that reduction in size of the heart at the end of the first week was very little although the œdema had disappeared. At the end of the second week only, the heart regained its normal size. Single pictures would only give a rough idea of the enlargement of the heart. Only the successive pictures of the same patient would show the actual reduction in size. In Case 5, the heart would have been declared normal but the second picture after treatment showed that the heart was really enlarged and definite reduction in size was observed after treatment.

Summary

1. Teleroentgenography of the heart in beriberi was studied in 15 cases from 28 plates.

2. The heart was enlarged in 14 (93·33 p.c.) of the cases.

3. The enlargement was observed in all the chambers in the following order: (*a*) right ventricle, (*b*) right auricle, (*c*) left ventricle and (*d*) left auricle.

4. Treatment either by vitamin B_1 or by liberal diet reduced the size of the heart in 10 of the 11 cases. The actual reduction in size could not be observed in the remaining 4 cases, since a single picture only was taken.

5. Five cases showed prominent pulmonary conus and reduction in the size of the conus was observed in 3 cases after treatment.

6. Two cases showed prominent superior vena cava and the shadow disappeared in both the cases after treatment.

7. One case showed congestion of the lungs as a result of heart failure.

Acknowledgment

My thanks are due to Dr. P. Kesavaswamy and Dr. C. Benjamin for the skiagrams.

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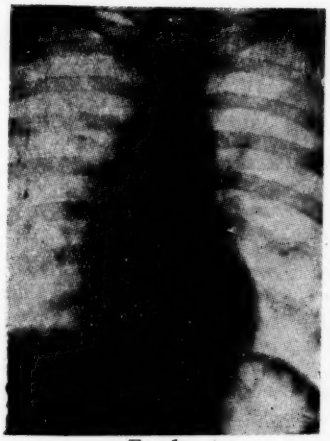

FIG. 1

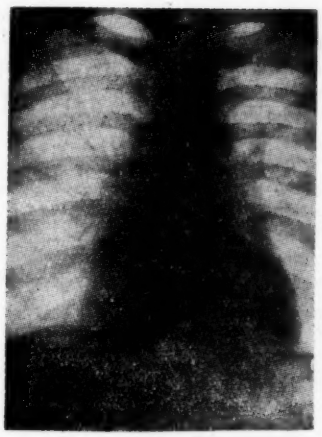

FIG. 2

FIG. 3

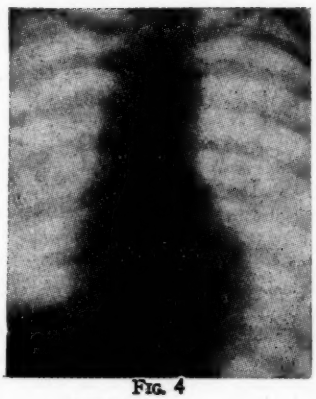

FIG. 4

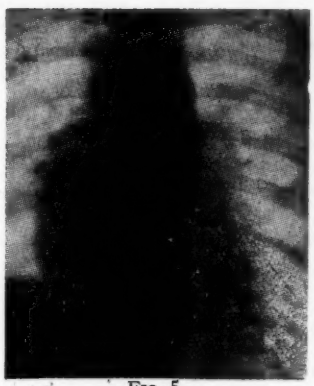

FIG. 5

FIG. 6

FIG. 7

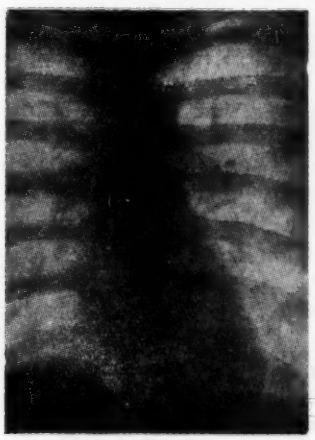

FIG. 8

EVALUATION OF SODIUM BISULPHITE AND ASCORBIC ACID AS ANTIDOTES OF COBRA VENOM

FAZLE KARIM KHAN, M.B., B.S.
From the Pharmacology Department, Osmania Medical College, Hyderabad, Deccan

The problem of the treatment of cases of snake-bite is still not fully solved. Attempts are, therefore, made to find out antidotes.

Ghosh, De and Chaudhuri (1941) report, that they kept solutions of the neurotoxin of the common Indian cobra and different reducing agents in test-tubes for 24 hours, and injected the 24 hours' old solutions into pigeons. They obtained a loss in the toxicity of the neurotoxin, as a result of its contact with the reducing agent for 24 hours. Of the compounds used, sodium bisulphite caused the greatest loss in toxicity *viz.*, 95·2 per cent while ascorbic acid caused 8·6 per cent.

I tried these compounds *in vivo*. The animals used were dogs. The animals were caught from the streets a day or two before the experiment and kept in cages on ordinary diet and water till the hour of the experiment. I adopted this procedure to imitate nature.

The venom used was a sample, kindly supplied by the Director, Haffkine Institute, Bombay, more than a year ago. It has always been kept on strong sulphuric acid in a desiccator. Bannerman (1914) found out that 0·25 mg. of the dried cobra-venom is the m.l.d./Kg. for the dog. During some experiments carried out by me in 1941, I found that all dogs do not die with 0·25 mg/Kg. I, therefore, gradually increased the dose, till I found that of the sample of venom in stock, 0·6 mg. per Kg. was the m.l.d. By m.l.d. I mean the quantity that will kill the animal with certainty. This determination was necessary before pronouncing the efficacy or otherwise of any supposed snake-bite cure.

0·6 mg/Kg. of this venom was injected into the muscles of the lower third of the medial aspect of the left thigh, this being the site used by me for an intramuscular injection of the venom. The injection was made intramuscularly because when we compare the length of the fang of an adult cobra with the thickness of the skin, subcutaneous tissue and the deep fascia, we conclude that in the case of a good bite, the fangs always enter the muscles.

The total amount of the m.l.d. used in these experiments ranged from 4 mg. to 8 mg.

The antidotes used were sodium bisulphite (Merck) and ascorbic acid (Bayer). The amount used was 500-1000 mg. The injections were made intravenously. The interval between the injection of the venom and the injection of the reducing agent was between 5 and 10 minutes.

The result was that each and every dog died, with the development of all the usual symptoms of cobra venom poisoning.

Conclusions

1. Sodium bisulphite and ascorbic acid failed to prevent one surely lethal dose of cobra venom, from exerting its toxic influence on the nervous system of the dog.
2. The intensity of the symptoms was the same, as that observed in dogs which were not given these antidotes.
3. The interval between the injection of the venom and the death of the dog was not reduced by these antidotes, to any extent.
4. The snake venoms are known to resemble the bacterial toxins in their pharmacological behaviour. Bacterial toxins are neutralized by the antibodies contained in the respective sera. Any compound which neutralizes a bacterial toxin *in vivo*, may neutralize a snake venom *in vivo*. As long as such a compound is not known to science, the antivenomous serum remains the only potent antidote.

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NOTICE

The following resolution was passed by the XIX All-India Medical Conference held at Patna during the last Easter holidays:—

> "In order to stimulate intensive study and research on Puerperal Sepsis the control of which is of vital importance to the nation, this Conference recommends to various medical institutions in the country to undertake detailed investigations of this disease in its different aspects, bacteriological and clinical, and to make available to the profession their findings in this respect. This Conference recommends to the Indian Medical Association to offer a medal or prize for the best paper on the subject to be read at the next session of the All-India Medical Conference."

Dr. K. S. Ray, an Ex-President of the Indian Medical Association, has offered a Gold Medal for the best paper on the subject to be read at the next session of the Conference to be held at Ahmedabad during the ensuing Christmas holidays. The last date for receipt of papers will be notified later.

P. B. MUKERJI,
Hony. General Secretary,
Indian Medical Association.

TREATMENT OF ANÆMIAS WITH SPECIAL REFERENCE TO IRON THERAPY

N. S. VARIAVA, M.D., M.S. (BOM.),
Honorary Physician, G. T. Hospital, and Teacher of Clinical Medicine, Grant Medical College, Bombay.

Introduction

The scope of iron has been very much narrowed due to advancement of our knowledge of anæmias. Iron is no longer used empirically in all cases as was done a few years back. It is now used rationally and with good effect in a few of the anæmic conditions.

There is a group of anæmic conditions in which deficiency of iron is the main underlying cause. This deficiency may result from intake of insufficient quantities of iron into the system, or as is more common, from lack of utilisation of iron. Iron is essential for the proper formation of hæmoglobin. Its specific effect consists in increasing the number of red blood corpuscles, and the amount of hæmoglobin, if they are deficient, such as is found in simple anæmia, chlorosis, amenorrhœa, cardiac disease, nephritis, syphilis, tuberculosis, recovery from hæmorrhage, malarial cachexia, and in convalescence from acute diseases.

The chief source of iron is food. A small amount of endogenous iron also results from breaking up of old red blood cells, and the disintegration of their hæmoglobin. In the food, iron is taken both in organic as well as inorganic forms. For a long period the controversy was going on whether inorganic iron is absorbed from the intestinal tract. It has now been definitely proved that inorganic salts of iron can be absorbed from the intestinal tract.

Iron is absorbed by the epithelium of the duodenum and the small intestine. The leucocytes carry the iron through the mesenteric lymphatic glands, or directly by the blood, first to the spleen, and then to the liver, where it is stored and gradually elaborated into complex organic iron compounds such as "ferratin". Thus it is important to remember that liver is not only a store-house of iron, but in it are also built up complex organic iron compounds. From the liver organic iron compounds are taken by the blood to the bone marrow as required. Here it forms hæmoglobin. Within the red cell iron exists only as hæmoglobin. Surplus iron is excreted through the mucous membrane of the large bowel.

For the normal development of red blood cells of proper size and shape, as well as for the deposition of hæmoglobin in the red cells in sufficient amount, iron is very essential. Iron is helped in its action by small traces of copper, manganese, calcium, thyroxin, vitamins A, B, C and D. How these various substances act is very imperfectly understood. They definitely help its absorption from the intestinal tract, and after absorption in its proper utilisation, for the formation of hæmoglobin.

As said above, iron deficiency would result from inadequate quantities of iron in the system. The normal iron-content of the body is about 3 to 3·5 grams of iron, of which about 2·4 to 2·7 grams are in the form of hæmoglobin. (Ghosh, 1930). Deficiency of iron soon leads to the development of anæmia. Iron deficiency anæmias can thus result from:—

(1) Deficient intake of iron in the food, (2) imperfect absorption of iron from the intestine, (3) interference with the storage of iron in the liver, (4) defective utilisation of iron by the bone marrow, and (5) deficiency of copper, manganese, calcium, thyroxin, vitamins A, B, C, and D.

Besides this, there is another group of anæmic patients in whom iron is also helpful. In this group there is no defect in the absorption of iron from the intestinal tract, or after absorption in the proper utilisation of iron. These anæmias are produced because the circulating blood is depleted in quantity or in quality through hæmorrhage, or other causes. In malaria for example, the red blood cells are destroyed by malarial parasites; a person may become anæmic due to repeated bleeding from piles.

Classification of Anaemias where Iron is Indicated

Anæmias where iron is clearly indicated can be classified into three distinct groups: (1) true iron deficiency anæmias, (2) hæmolytic anæmias, and (3) post-hæmorrhagic anæmias.

True Iron Deficiency Anæmias—

(A) Primary: (*a*) idiopathic hypochromic anæmia, and (*b*) chlorosis.

(B) Secondary (there is a recognisable defect):

(*a*) Deficient intake of iron in the food: (*i*) prolonged starvation, (*ii*) too rigid dietary in the treatment of peptic ulcers, (*iii*) nutritional anæmia of infants, and (*iv*) nutritional anæmia of pregnancy.

(*b*) Imperfect absorption of iron from the intestine: (*i*) chronic diarrhœas, (*ii*) sprue, (*iii*) cœliac disease, (*iv*) idiopathic steatorrhœa, and (*v*) catarrhal conditions of the intestinal tract.

(*c*) Defective utilisation of iron by the bone marrow—hypochromic anæmia associated with:—(*i*) malignant disease, (*ii*) tuberculosis, (*iii*) syphilis, (*iv*) infections.

(*d*) Deficiency of copper, manganese, calcium, thyroxin, vitamins A, B, C, and D—hypochromic anæmia found in:—(*i*) scurvy, (*ii*) pellagra, (*iii*) myxœdema or cretinism, and (*iv*) rickets.

Hæmolytic Anæmias (red blood cells are destroyed or broken up within the body by some toxic substance)—

(*a*) infective fevers, (*b*) malaria, (*c*) blackwater fever and (*d*) paroxysmal hæmoglobinuria.

Posthæmorrhagic Anæmias (resulting from chronic hæmorrhage)—

(*a*) hæmoptysis, (*b*) hæmatemesis, (*c*) bleeding piles, and (*d*) menorrhagia.

Reviewing the classification we find that in the first group the anæmia has resulted from primary lack of iron in the system whereas in the second and third groups the lack of iron is secondary to red blood cell destruction.

ROLE OF IRON IN THE TREATMENT OF ANAEMIA

There are several preparations of iron available in the market. Even in the B. P. there are a number of iron preparations. It is to be decided which of the preparations are to be used and how.

In whatever form iron salts are taken by the mouth, they are mostly converted into ferric chloride in the stomach. It was believed that an albuminate of iron was formed in the stomach, but this is incorrect. Even iron albuminate is decomposed into the chloride. If the iron salts are given in excess, or if gastric juice is deficient, all the hydrochloric acid of the gastric juice will be used up in combining with iron salts to form the ferric chloride. Thus digestion will be impaired. Moreover, if strong acid salts of iron are administered, iron will combine with hydrochloric acid liberating the strong acids. These will definitely irritate the gastric mucous membrane. When the contents of the stomach go to the doudenum, ferric chloride is converted into an oxide of iron. This remains in solution in the intestinal canal, because of the presence of organic matter in the fluid. The iron salts are then absorbed by the epithelium of the duodenum and the small intestine. The unabsorbed part passes to the large intestine. Here sulphur compounds, tannic acid (derived from vegetable food), nascent hydrogen, and other readily oxidisable products of decomposition are present. They combine with iron and form ferrous sulphide and tannate. They are then passed out in the fæces which is coloured black.

This brings us to three important considerations: (1) Should hydrochloric acid be used as a routine in the treatment of anæmias? (2) What salts or preparations of iron should be used? (3) Should iron therapy be given alone, or be supplemented by substances like liver, copper, manganese, calcium, thyroid, and vitamins?

THE ROUTINE USE OF HYDROCHLORIC ACID

Davis and James (1930) have shown that a high percentage of perfectly healthy persons in the later stages of life secrete no hydrochloric acid in the stomach. They found that 32 per cent of normal healthy persons over 60 years were achlorhydric. If however, a preliminary injection of histamine was given before the test-meal, the figure was reduced to 15 per cent. Witts (1930) has shown that achlorhydria may be associated with primary (pernicious), or secondary (simple achlorhydric) anæmias. The latter is a common condition. It is present even before the development of the anæmia, and persists even though the anæmia is cured. This anæmia is always of hypochromic microcytic variety. These observations led Hurst and other workers to recommend the use of hydrochloric acid in anæmias, particularly in pernicious anæmia. The rationale of prescribing large doses of hydrochloric acid was the sterilisation of the upper gastrointestinal tract, since the gastrointestinal flora were supposed to be the underlying cause of pernicious anæmia, and other secondary types of anæmias. But this theory now no longer holds good. Witts (1931) has shown that hydrochloric acid has no effect in the treatment of simple achlorhydric anæmia, nor does it enhance the value of iron therapy. The conclusion that the majority of workers have come to, is that the hydrochloric acid therapy is of little value, except in the treatment of gastrointestinal disorder. Hydrochloric acid is a valuable drug in the treatment of vomiting and dyspepsia, especially in the flatulance and diarrhœa which sometimes accompany the achlorhydric state. Drachm one of hydrochloric acid well-diluted with water, and flavoured with orange juice often works like a charm. Wilkinson and Brockbank (1931) believe that better results are obtained if pepsin is given in addition.

PREPARATIONS OF IRON TO BE USED

Iron salts can be classed into four different groups: (1) ferrous salts, (2) scale preparations, (3) ferric salts, and (4) organic iron. The choice of a preparation from amongst these salts becomes a difficult problem for a young practitioner. He should choose a preparation which has the maximum of theraputic effects, with a minimum of deleterious effects. He should select a preparation which could be used effectively in the smallest possible dose. He should use a preparation which is nonastringent. Organic salts of iron are nonastringent. Of the inorganic salts, ferrous salts are less astringent than ferric salts.

The theraputic use of iron is reviewed by Witts (1931). The preparations of iron in order of efficiency are: (1) ferrous salts, (2) scale preparations, (3) ferric salts, and (4) organic iron. Majority of workers prefer either ferrous salts, or ferri et ammon citras. The advantage of ferrous sulphate over ferri et ammon citras lies in the fact that the former has a much smaller dose as compared to the latter. According to Witts (1931) "the ferrous salts of iron are active in smaller doses, and produce a more rapid effect than any other preparation of iron." Ferrous sulphate is described as "by far the most effective source of iron in the treatment of iron-deficiency anæmias" (Parsons and Hawksley, 1933). The small dose of ferrous salts usually necessary for maximal results obviates most of the unpleasant effects of large doses of other iron salts. In the opinion of most of the workers the best way to administer iron is to use it as ferrous sulphate. Exsiccated ferrous sulphate is still better in every way than ferrous sulphate.

Ferrous salts are most commonly used in two forms: (1) ferrous carbonate usually in the form of pil. ferri (Blaud's pill), or (2) as exsiccated ferrous sulphate usually in the form of some patent preparation. Another iron preparation that is very commonly used

is ferri et ammon citras. The latter is usually prescribed in a mixture form. Of the three preparations the best one to use is exsiccated ferrous sulphate.

Blaud's pill is given in doses of grains 5, three times a day, after meals. Ferrous sulphate is prescribed in doses of grains 5, while exsiccated ferrous sulphate in doses of grains 3. Ferri et ammon citras should be used grains 10 to 15 per dose. As iron is absorbed slowly from the intestinal tract, three doses per day is sufficient. It must be given after meals only. This dose is often sufficient to raise the rate of hæmoglobin regeneration 1 per cent daily, which is considered optimal.

In severe cases, the treatment should be started as above, but after a few days the dose must be increased. Witts (1931) recommends that the minimal daily effective dose should be at least twice the pharmacopœial dose, *i.e.*, Blaud's pill grains 30, exsiccated ferrous sulphate grains 20, and ferri et ammon citras grains 60 per day.

In many cases in spite of such heavy doses, the improvement is not manifest for a few days, may be even for one month or so. The treatment should be continued for about two months, and then the maximum benefit will be observed. The dose can then be reduced to the usual suggested above, and should be continued for a month or more as necessary. Then a maintenance dose of iron may be continued to prevent a relapse which must be at least one-third of the therapeutic dose and should be given once or twice a week.

Ferri et ammon citras is usually given in mixture form. The ferrugenous taste of iron may be masked by giving it with syrup aurantii.

There are certain points which must be remembered during the administration of iron. These are: (1) Iron preparations should be given after meals. (2) If the iron treatment is to be a prolonged one, the drug should be stopped at intervals of a few days. (3) Iron always causes constipation, so at regular intervals purgatives, are to be given, *e.g.*, salines, or it may be combined with a purgative, *e.g.*, pil aloes et ferri. (4) Iron often causes gastric upset and headache when it should be stopped for a few days. (5) Iron preparations stain the tongue and teeth black. The best way to prescribe iron then is in the pill form. But it is often found that when pil. ferri (Blaud's pill) is prescribed, the whole pill passes out, i.e., it is not easily disintegrated in the intestine. So iron pill is to be prescribed in such a form that it readily disintegrates in the intestine. For this reason it may be sugar-coated, or put in karatin capsules.

Iron should not be given by injections, owing to the long duration of the treatment, and the considerable difficulty of giving adequate doses by this route (Witts, 1931).

Supplementary Treatment with Liver Extract, Copper, Calcium Salts and Vitamins

Very frequently we come across cases of microcytic hypochromic anæmia, where in spite of very extensive iron therapy, we do not observe satisfactory reticulocyte response. In such cases we may have to supplement iron therapy with substances like liver, copper, manganese, thyroxin, calcium salt and vitamins. But there are certain indications for the use of these various substances.

Liver and Secondary Anæmias—There are many cases of hypochromic anæmia which do not respond to iron treatment alone, or to liver treatment alone, but when liver and iron therapy is combined they show marked improvement. Several workers have stated that the liver therapy enhances the value of iron. Liver should be given orally as a part of the diet, which should be rich in factors known to be valuable in hæmoglobin manufacture. Whole liver and iron orally was found to be more beneficial than liver extract injections and iron. The combined treatment is particularly useful in the treatment of chronic hæmorrhagic type of secondary anæmia. Other indications for the combined treatment are, anæmia due to hookworm, anæmias associated with undernutrition, dysentery, and pregnancy.

Copper and Manganese in Hypochromic Anæmias—Mills (1930) found a number of cases of secondary anæmia where liver and iron treatment showed no improvement. This is most commonly found in idiopathic hypochromic anæmia. He treated these cases by combining copper and manganese with iron. Prompt improvement occurred in all cases. The blood count was restored to normal. Elvehjen (1932) has shown that in the absence of copper and manganese, iron, whether in organic or inorganic form, is ineffective for the cure of nutritional anæmias. Lewis (1931) found that cure resulted much more quickly, in weeks rather than in months, when copper and manganese were combined with iron.

Copper and manganese are needed in small traces only. Mills used copper and manganese sulphate in doses of 0·01 grain each. How these small doses act is not known. They probably act as catalysts. They accelerate the absorption of iron from the intestinal tract. They also are necessary for the proper utilisation of iron for the synthesis of hæmoglobin.

Thyroxin and Secondary Anæmias—In persons with normal thyroid gland, the importance of thyroxin in blood regenerations is not evident. But when the thyroid secretion is deficient as in myxœdema, its important role in the production of anæmia at once becomes manifest. In myxœdema the anæmia is of the mycrocytic hypochromic variety. No amount of iron therapy will improve the anæmia. But if the patient is put on the thyroid extract side by side with iron, the improvement is rapid. Thyroxin has a marked influence on body metabolism, and iron metabolism is a part of the general body metabolism. Thyroxin helps in absorption of iron from the intestine, and after absorption in the utilisation of iron for the synthesis of hæmoglobin (Mayerson, 1933).

Calcium and Iron—Like thyroxin the role of calcium in the causation of anæmia is not clearly appreciated. But its importance manifests itself in conditions where there is lack of calcium as well as iron. In conditions like rickets, osteomalacia and nutritional anæmia of

pregnancy and childhood, it is found that the anæmia will not improve with iron, unless calcium in adequate amount is administered at the same time. Calcium lactate grains 20, three times a day should be given with iron. How calcium acts is not known. Thompset (1940) suggests that the absorption of both iron and copper from the alimentary tract is influenced by the gastric acidity, and the calcium contents of the diet. Calcium is an "iron sparer", *i.e.,* an element which promotes the conservation of iron and its rapid replacement.

Anæmia and Vitamin Deficiency—Koessler *et al* (1926) investigated the relationship of anæmias, both primary and secondary, to vitamin deficiency, and they came to the conclusion that the vitamins had a certain important role in the formation and development of anæmias.

Vitamin A—About the role of vitamin A these authors came to the following conclusions: (1) Blood regeneration cannot take place without the presence of vitamin A. (2) Addition of vitamin A to the diet of animals long depleted of vitamin A reserve, brings about rapid formation of new blood cells. (3) Rate and intensity of blood regeneration is a function of the quantity of vitamin A added. This definitely proves that vitamin A has a marked influence on the cycle of development of the red blood cells; and the rate and intensity of blood regeneration depends on the store of vitamin A in the system.

Vitamin D—The clinical association of rickets and anæmia is well known. Reviewing the literature Crowley and Taylor (1939) conclude that rachitic children are often, but not always anæmic. They also report their own experience of iron-resistant anæmia associated with rickets in school children. They conclude that "from the results it is justifiable to state that mild iron resistant hypo- or orthochromic anæmia associated with rickets is to be found among school children. Low blood phosphorus is rapidly corrected by massive doses of vitamin D, but the anæmia is relieved only by a combination of iron and vitamin D. It appears that adequate supplies of vitamin D are essential in these children if iron is to be properly utilised for blood formation." If such is the state of affairs in rich countries, what would be the position in India, where chronic malnutrition is prevalent in both adults and children. Numerous Indian observers have pointed out the prevalence of anæmia in mothers and children in working class people. Pregnant women and nursing mothers even in middle class families in India suffer from anæmia to a greater or lesser degree. The ideal treatment for these would be to provide vitamin D in sufficient dosage together with iron.

Vitamin B—Vitamin B is not a single substance, but is made up of a number of different substances. As all the different fractions of vitamin B complex are not known to us, it is but natural that we do not know the exact role of vitamin B complex in the causation of anæmia. Vitamin B_1 brings about absorption of iron from the intestinal tract (Sparks and Collins, 1937). If vitamin B_1 is absent from the normal diet, anorexia is the commonest early symptom noted. This symptom soon disappears, and the appetite is markedly improved if adequate amounts of vitamin B_1 are added to the diet. Vitamin B_1 is particularly indicated in anæmias resulting from prolonged starvation, or in cases of nutritional anæmias of pregnancy and infancy. It is also indicated in anæmias resulting from gastrointestinal conditions, *e.g.,* idiopathic steatorrhœa, chronic diarrhœas, sprue, etc. Deficiency of vitamin B_2 (riboflavin) leads to changes in the mucous membrane of the mouth, and the gastrointestinal tract, with the resulting glossitis and gastrointestinal symptoms. As a result, interference with absorption of substances like iron, calcium, etc., follows. This soon leads to the development of microcytic hypochromic anæmia. Absence of P. P. factor (nicotinic acid) also causes gastrointestinal changes which leads to interference with absorption of antianæmic substances. When vitamin B_6 in the form of rice polishing extract was fed as a supplement to the diet, the anæmia was cured much more rapidly than is common for pernicious anæmia, and other nutritional anæmias (Fouts *et al,* 1938).

The most effective way of giving vitamin B is in the form of vitamin B complex. It not only contains the various fractions considered above, but many more unknown factors also, and thus its nutritional value is greater than any individual factor. Yeast is the best and the cheapest source of vitamin B complex. The cytochrome of yeast potentiates the action of iron and facilitates its absorption and utilisation. When yeast extract is administered in cases of sprue, and cœliac disease in children, the anæmia has improved marvellously. In addition to its antianæmic value, the striking response of yeast (vitamin B complex) is due in a large part to improved appetite, and assimilation, brought about by normal functioning of gastrointestinal tract.

Vitamin C—Parsons and Hawksley (1933) noted that red blood cell regeneration does not take place in the absence of vitamin C. He further noticed that when vitamin C is given with iron better absorption of iron takes place. Not only that, it also helps in better utilisation of iron for the synthesis of hæmoglobin.

Summarising the value of vitamins in anæmias Kœssler *et al* (1926) come to the following conclusions:—(1) Blood changes and changes in the gastrointestinal tract may be due to vitamin A underfeeding over a prolonged period. (2) Nervous symptoms might be related to absence or deficiency of vitamin B. (3) Tendency to hæmorrhages found in severe anæmias may be due to partial or complete lack of vitamin C. (4) A definite relationship exists between chronic vitamin deficiency, and certain anæmias. (5) Rationally balanced diet is most essential in blood regeneration of patients suffering from severe anæmia.

Blood Transfusion in Anaemias

There are cases of anæmias, where the amount of blood in general circulation is so much depleted, that unless the amount is restored back, the anæmia

will not improve with iron. Turner (1934) gives the indications for transfusion in anæmias, as follows:—Transfusion should be done in anæmias following—(1) severe primary hæmorrhage; it should be done in cases of shock resulting from hæmorrhage; he recommends that if the red blood corpuscle count is below four million, and the white blood count above 30,000, within six hours of the reception of the wound, blood transfusion should be done and in these cases the results are most satisfactory. (2) Secondary hæmorrhage. (3) In cases of secondary anæmia due to duodenal or gastric ulcers, with repeated attacks of hæmorrhage. (4) Hæmophilia.

Not more than 500 c.c. of blood should be transfused at each time. It may be repeated, if necessary, after a few days. The most important point to remember is that the blood transfusion is a lifesaving measure; it does not bring the blood to normal. The anæmia requires iron treatment.

Diet

The improvement will not be maintained unless proper attention is given to the diet of the patient. A well balanced diet rich in factors known to be of value in the manufacture of hæmoglobin is necessary. Such diets are liver, kidney, egg, and red meat. Plenty of green vegetables should also be eaten.

Prevention

We see from the above discussion that many of the anæmias are the result of inadequate, badly balanced diet. A large percentage of working class women and men partake of food which is very poor in the necessary constituents of diet. It is grossly deficient in animal protein and green vegetables. Such a diet if long continued, not merely produces an anæmia, but if the blood is lost through any channel, or if the red cell production is poor as a result of toxæmia or infection, it will fail to supply the necessary factors to make up the deficiency. Prevention, therefore, "lies more in butcher's and green-grocer's shop, rather than in the druggist's store."

Summary

(1) Digestion and metabolism of iron are discussed. (2) Indications for iron treatment, and the methods of administration in anæmic conditions are dealt with. (3) Importance of diet in prevention is stressed.

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ILIAC ABSCESS*

U. P. SINHA, F.R.C.S.E.,
Lecturer in Surgery, P. W. Medical College, Patna.

Introduction

Iliac abscess has got little attention in the text books of surgery and there are only scanty references about it in the literature. Yet this disease has been a bugbear to many of us in the beginning. It is just possible that we in Bihar are singularly unfortunate in having plenty of these cases. I would, however, like to know from my colleagues as regards the incidence of this disease in their respective tracts, especially from those who come from filarial districts.

What attracted my attention was the number of cases of iliac abscess in all stages—cases going downhill inspite of drainage, cases with persistent sinus or sinuses, cases of fæcal fistula were a common sight. I soon realised that a certain percentage of these cases were tuberculous in origin. The majority, however, was of non-tuberculous nature and by no means easily amenable to treatment.

Materials for Study

During the years 1938 to 1941, 84 cases of iliac abscess were admitted into our hospital. Out of these, 70 were males and 14 females. The preponderance of males in this collection is difficult to explain unless we take trauma as one of the causative factors. All the cases were treated by operative measures except three which cleared up by sulphonamide therapy alone. The analysis of these cases are given below in a tabular form:

Analysis of Cases Treated During the Period 1938-41.

Year	No. of Cases	Male	Female	Cured	Otherwise†	Dead
1938 ..	29	23	6	20	6	3
1939 ..	15	13	2	10	4	1
1940 ..	27	22	5	14	9	4
1941 ..	13	12	1	10	2	1
Total ..	84	70	14	54	21	9

† In this group are included cases that lingered and got complicated as also those which left against medical advice.

In short, out of the 84 cases 64·28 per cent were cured and 35·71 per cent went wrong (this figure includes the cases of death as well).

I would like you to consider two things in connection with the above figures. Firstly, the appalling number of cases that ended in failure *i.e.*, 35·71 per cent; and secondly, the period of hospitalisation. In this series patients have stayed in hospital from one to 117 days. The percentage of

* Read at the Scientific Section of the XIX All-India Medical Conference, Patna, April, 1943.

average hospitalisation works out to be 29·57 days, *i.e.*, roughly one month. Does it not make you ponder over the problem? Is it not worthwhile finding out ways and means by which we can ameliorate this condition? I am sure if we go into the causation and the method of cure it will be a great help to us and to those who come after us.

Clinical Aspect

Causative factors—As regards the causative factors I shall again refer to *trauma* which may be responsible for the preponderance of iliac abscesses in the male. I shall give another evidence which will perhaps support this view. Two of my own cases which clinically resembled iliac abscess proved on cutting open to be nothing but hæmatoma.

Tuberculosis. In cases of tuberculosis of the hip the tuberculous debris may perforate the floor of the acetabulum and present above the Poupart's ligament. This type of case is not uncommon in my experience. Clinically, it is very difficult to diagnose, especially in early cases. Flexion deformity due to psoas spasm is present in all cases of iliac abscess, whether of tuberculous origin or not. Wasting of the thigh to some extent is also present in all cases of iliac abscess. The limitation of movement in all directions which is characteristic of tuberculous hip is a late phenomeon and may not be present. I was consulted by a colleague of mine for a boy suffering from iliac abscess. Clinically, the hip joint was free and I confidently declared it to be a case of pyogenic origin. Subsequently, I operated on the boy. Creamy pus came out. Sinus persisted and two months after, well marked erosion of the hip joint was seen in the skiagram. After having fallen into trap once, one naturally feels like getting his cases x-rayed as a routine measure before operation. This on the whole works well but is not a foolproof method. In one of my cases the radiologist's report was negative, but subsequently it turned out to be a case of tuberculous hip. The lesion might have been a very early one or of synovial type.

I would, therefore, suggest a double check method, as a routine practice. Firstly, the pus to be aspirated and cultured and secondly, the hip joint x-rayed.

Pyogenic infection—A large majority of cases are of pyogenic origin—perhaps of metastatic infection. The site is determined by trauma factor. In our series of cases both streptococcus hæmolyticus and staphylococcus aureus were found.

Filaria—Bihar is a filaria-infested province. Many suffer from filariasis although symptoms may not be present. How far filaria is responsible for an iliac abscess I am not in a position to state.

Diagnosis

Iliac abscesses are situated above the Poupart's ligament and are parallel to it. Appendicular and psoas abscesses are easily differentiated.

Treatment

Liberal incision parallel to and above the Poupart's ligament is the secret of success. In the case of a small abscess

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MEDICAL PREPAREDNESS SERIES

The war coming on to the borders of India has revealed to us all the terrible aspects of the menace and it has become apparent that the profession shall have to take up quite a large share of responsibility in A.R.P. measures. A series of articles on war medicine, A.R.P. and allied subjects is being published with the idea of helping the cause of necessary medical education. This is the third article of the series.—Editor, *J. Indian M. A.*

AIR-RAID PRECAUTIONS AND MEDICAL MEN

K. S. RAY, M.A., B.SC., M.B., CH.B. (EDIN.), M.L.C. (BENGAL)
Calcutta.

The fact that our medical curricula do not include problems that may arise during an actual air raid, was recognised by the Indian Medical Association with the beginning of the war. During the civil war in Spain it became apparent to the members of the Indian Medical Association that in future, intensive and extensive air-raids will play the the main role in future wars. The pact made by Japan, Germany and Italy left no doubt in our minds that India will be in great difficulties if she does not prepare to face war in all its aspects. The Indian Medical Association, therefore, passed the following resolution at its Annual Conference in 1938 at Meerut.

"In view of the fact that in future, wars are not going to be confined to the fighting forces alone but would affect the civil population as well, this Conference requests the Central and Provincial Governments to institute a survey of all the available medical resources in their respective provinces which can be requisitioned at short notice to give relief to the civil population in cases of emergency arising out of air-raids and other eventualities and to give facilities to medical practitioners to obtain necessary training in this regard.

"This Conference further requests the Indian Medical Association to lend its whole-hearted co-operation to the Government in making the survey and the facilities offered for training a success and draws the attention of the Red Cross Society and other similar organisations."

This was reiterated in 1939 in the following words:

"In view of the havoc and destruction which the civil population is exposed in modern warfare and the possibility of such happenings in India, this conference is of opinion that medical practitioners should obtain the necessary training in the prevention and treatment of poison gas and air-raid casualties, and should organise themselves to meet such emergencies or to render help to the organisations already existing for the purpose."

This was again reiterated at the next year's conference. It was circulated and the general medical public was made interested in the subject. Both the medical and the non-medical public showed so much interest in the subject that the Indian Medical Association thought it proper to impart basic knowledge on air-raids both to the medical and lay public. To these ends it published in 1939 two booklets—"Treatment and Prevention of Poison Gas" and "Air Raid Precautions (Householder's guide)". These were published long before any other Indian hand-book was published on

the subject. The following extracts from the preface of these booklets will give us the actual position at that time:

"In countries which are considered to be exposed to air-raids, a series of Air-Raid Precaution hand-books have been published by their respective governments. These hand-books describe the measures for safe-guarding the civil population against air attacks and they have become a necessary part of the defensive organisation of every country vulnerable to such attacks.

"The need for these hand-books arises from the fact that an attack from the air, however remote it may be, is a risk that cannot be ignored and because preparations to minimise the consequences of attack from the air cannot be improvised on the spur of the moment but must be made, if they are to be effective, in time of peace.

"In response to insistent demands from the branches and the medical profession for authentic information regarding treatment and protective measures against air-raids, this pamphlet is published under the authority of the Indian Medical Association." These booklets are still available for public sale.

The effect was almost electrical; the various branches of the Indian Medical Association organised lectures, symposia and other forms of general dissemination of knowledge in air-raid problems. Courses in first-aid, for the medical and lay public were organised and special numbers of various medical journals began to appear. This also gave a filip to organise voluntary and subsidised first-aid posts. Both medical and lay public took up the work in right earnest and constructed a country-wide organisation. Happily, the Government could be made to understand the importance of this voluntary movement of the medical men and they took up the work in an organised fashion.

At this stage, with the entrance of the Government in the field with a much more elaborate scheme, it was felt that some sort of remuneration should be offered to the medical volunteers. Committees were set up in the different cities, which were in constant touch with the Government and contributed quite a large share in the final formation of the existing schemes. At the present moment many provinces have got regular Emergency Medical Services Scheme A perusal of these schemes will reveal at least two chief drawbacks. The most important of these is the entire lack of uniformity amongst them. Problem remaining same, it does not behove that the remedies should be different and the Indian Medical Association is trying to get the opinion of all medical men of India through its various branches on this point. The second point is that the schemes are not drawn on any calculations based on the experiences of extensive air-raids on London, Rangoon and other places. Much improvement in these schemes is possible and shall have to be made if they are intended to serve the purpose aimed at. The Indian Medical Association has suggested improvements from time to time but very little has been done so far.

While Government is busy with schemes and amendments, we, medical men, must not forget that (as a profession, which is able to render the greatest help at the time of need) we have certain responsibilities towards the medical preparedness of the country. Those of us who are working in the first aid centres, should keep their personnel up-to-date both in knowledge and practice by constant lectures discussions and staging of mock accidents; while hospitals should be run for a certain time, say two hours a week, as a model "air-raid hospital". Those medical men who did not, for various reasons, attach themselves with any of these organisations, should gather some knowledge on air-raids, damages done by blast and other allied subjects so that they can offer immediate help to their own household and their neighbours at the time of actual air-raids. We would like to point out again that repeated lectures, discussions, demonstrations and mock trials are the known sure ways of preparing for such situations. Unit by unit, person by person, should know his or their duties and must know where he comes in in the whole scheme, so that he shall know exactly where he is to go and what he is expected to do, while the siren goes. The efficiency of the work to be done at the time of actual air-raids will naturally depend on these preliminaries.

Lastly, we would point out that during air-raids, both the military and civil population will be affected as was seen during the last air-raids and as a member of a noble profession, we must come out to treat the air-raid victims without any distinction of caste, colour or creed.

(*Continued from page 319*)

the surgeon has no other alternative but to give an incision in the anterior wall of the abdomen. Is bigger abscesses extending anterolaterally one may give an anterolateral or an anterior incision and a lateral counteropening. But however efficient drainage one may provide, the fact remains that in the recumbent position the iliac fossa remains the most dependent part. In a certain percentage of cases the stoma closes quicker than the filling up of the cavity and a sinus results. Fibrosis of the tract takes place. This I consider a calamity of the first rank. The surgeon has to scrape the cavity. The large gut is in the immediate neighbourhood which might also be scraped, or it spontaneously gets adherent to the track of the sinus and later opens into it resulting in a fæcal fistula. At present I have one such patient in my ward on whom three operations have been done to close the fistula but all have resulted in failure. The last one was done after a temporary cæcostomy, with the same result.

After this sad experience in several cases I started a continuous irrigation of the abscess cavity with normal saline by drip method and I have been fully rewarded. This stops stagnation of purulent discharge at the bottom of the cavity and helps in quicker healing. No more sinuses have resulted since.

Complication

I shall just touch on one complication. Three of my cases died of *secondary hæmorrhage* Sneezing or coughing may be enough to lead to it. Packing is ineffective. Tying of the iliac artery above is difficult and the patient dies. In a case where the pus is pointing, one has to be wary. When the pus has corroded the three layers of the musculature it has also done enough damage to the unsupported vessel walls which are literally bathed in pus. It is no wonder that the external iliac gives way.

SPECIAL ARTICLE

THE ROLE OF THE OBSTETRICIAN AND GYNAECOLOGIST IN FUTURE INDIA*

BAMAN DAS MUKHERJEE, M.R.C.O.G. (LOND.),
Calcutta.

ॐ

"वागर्थविवसंपृक्तौ वागर्थप्रतिपत्तये।
जगतः पितरौ वन्दे पार्व्वतीपरमेश्वरौ॥"

LADIES AND GENTLEMEN, I consider it a great honour and privilege to be invited to preside over the Obstetric and Gynæcological Section of the All-India Medical Conference and I thank you very much for having called me to this office.

I have on more than one occasion dealt with the various problems which we, as obstetricians and gynæcologists, are called upon to face every day in the course of our professional career. These problems are so vast and their solution requires so much close attention and endeavour that I cannot help repeating what I have said on different occasions and at different places.

Let me begin by saying that the day to day problems with which we are confronted in this unfortunate country of ours are peculiarly our own. In order to understand them in their proper perspective we have to take stock of conditions, political, economic and social which are peculiarly Indian and which, let me tell you at once, no one can understand better than the Indian medical practitioner. The workings of the mind of the Indian patient, her mode and manner of life, her dietary, the social influences and the environments amidst which she lives, moves and has her being, the economic factor resulting in deficient nourishment, the insipid climate, intercurrent diseases like malaria, tuberculosis, helmenthiasis—all these and many more are problems peculiar to the Indian soil and have to be carefully considered in appraising the present helpless state of the Indian womanhood and also in devising ways and means for bettering the lot of the Indian women in future.

Ladies and Gentlemen, the difficulties in our special sphere of gynæcology and obstetrics seem at first almost insurmountable and may well lead one to be pessimistic about their solution. But I am convinced that with a correct appreciation of these difficulties and a determined and concerted effort at their solution we can go to a great way in building up a bright future for our unhappy land.

The first and foremost difficulty which stares us in the face and baffles all our best endeavours towards ameliorating the condition of the Indian mother and child is the abject poverty of the people. The appalling morbidity and mortality amongst our women and children is, as you know, primarily due to the very low economic condition of the masses. No doubt it is a factor beyond our jurisdiction—a factor which the State has to tackle. But until this is done all our best wishes for a happy future for our mothers and sisters will be of no avail. It is idle to think of achieving any appreciable good until the standard of living of the average mother and child is raised considerably above its present level. The apathy of the State and, I am constrained to say even of our own men at the helm, is almost criminal.

Out of this poverty arises the utter ignorance and superstition of the Indian mother and prospective mother. Here, however, we cannot altogether plead want of jurisdiction. We can and we ought to do our very best to kill this monster of ignorance and superstition if we intend to achieve any solid results in the field of Maternity and Child Welfare. Women are inherently conservative and traditionally slow to move. A great deal, however, in my opinion, can be done by us to counter this ignorance and superstition if we act in concert. We can help greatly in spreading knowledge amongst our masses by circulating in popular provincial languages and by means such as the lantern slides, posters, placards, radios and other modern devices much valuable information regarding antenatal and postnatal matters and matters concerning maternity and child welfare in general. It is almost a truism to say that if we are to succeed in doing good to our community, Indian mothers, particularly of the rural areas, must have some intelligent appreciation of our work, specially antenatal and postnatal work. Not to speak of the rural women even our so-called educated mothers and prospective mothers are singularly lacking in the knowledge of domestic hygiene, infant-rearing and child-welfare. We have also, therefore, to consider the question as to how far it is possible to provide in the curricula of our girls' schools or women's colleges subjects such as Domestic Midwifery, Maternity and Child-Welfare.

Not the least important again of the problems confronting us is the problem of the Indian *dhai*. I need not recapitulate the terrible results of the inefficient handling of maternity cases by our *dhais*. They are, it is well known, invariably untrained, unclean and steeped in ignorance and superstition and are, moreover, recruited from the lowest strata of the society. This institution of *dhais* has definitely to go before millions of mothers and children can be saved from much preventible suffering, nay even death. I confess, this problem of *dhais* as not easy to solve in the first place because of the age-old tradition which makes the *dhai*, so to say, indispensable at childbirth and, secondly, because of the dearth of properly trained nurses and midwives. In many modern countries it has been found necessary to have recourse to legislation for the regulation of the practice of midwives. We have to consider how far legislation on similar lines is called for in this country and whether, at least for the present, we can compel the *dhai* to have some training in the art she practises before she is allowed to handle maternity cases. This institution of *dhais* will however persist until a large number of trained Indian nurses is made available to the average mother and until we succeed in having a network of maternity clinics and hospitals throughout the length and breadth of our country. Although things in the urban areas have improved a little yet the profession of nursing, particularly child-bed nursing, is not at all popular with our girls. It is necessary that facilities should be provided so that Indian girls of the better classes may be attracted to this profession. I cannot too strongly emphasise the needs of

* Presidential Address delivered at the Section of Obstetrics and Gynæcology of the Scientific Sessions of the XIX All-India Medical Conference, Patna, April, 1943.

hundreds of *sevikas* and nurses for our work. Training classes for our girls amidst better conditions and surroundings should be opened in every maternity hospital, clinic, girls' school and even by private practitioners throughout India.

We as obstetricians and gynæcologists cannot overlook the role in relation to maternity and child-welfare which, in the present day scheme of things, is played by the general medical practitioner in the mofussil area. Most intricate obstetric and gynæcological cases have to be attended to by him. We have in India a microscopic minority of practitioners trained specially in obstetrics and gynæcology. The result is that in rural and even in urban areas the general medical practitioner has of necessity, to bear enormous responsibilities which are out of proportion to his knowledge and skill in science and art of midwifery. It is, of course, no fault of his that he is called upon to shoulder this responsibility which, in the fitness of things, should devolve upon the obstetrician and gynæcologist. The fault lies in the system in the mofussil which denies him opportunities of gaining knowledge in the sciences concerned in the course of his practice. Midwifery has made rapid strides in the course of about the last 25 years. The general medical practitioner in our rural areas has no scope of having knowledge of these advances in science. I have suggested in the past and I repeat that, we should, as a body consider how far it is possible to give periodical facilities in the city hospital and clinics to the general medical practitioner practising in the districts by providing revisional courses in obstetrics and gynæcology. But the main thing necessary is a thorough revision of the hospital system now in vogue whereby non-service medical men are kept out of existing hospitals in the districts. In my opinion a healthy co-operation between the service men and the non-service men in the mofussil is a crying necessity. It is really by throwing open the gates of the State-managed mofussil hospitals to the general medical practitioner that we can improve his store of knowledge and experience and make him more fit to deal with very many maternity cases which must of necessity at present come to him.

A growing phenomenon which is engaging our attention to-day is the over-crowding of obstetricians and gynæcologists in the few cities in India, like Calcutta, Bombay and Madras. While, in the rural areas we have the ghastly spectacle of infinite havoc wrought amongst mothers and expectant mothers by the *dhais* and the ill-equipped general medical practitioner in the urban areas, we have the none too elevating sight of the qualified young specialist struggling for years to make a bare living and wasting his thought and energy over the problem of making both ends meet! While at present the rural areas offer no prospects to a man of special knowledge and skill and no scope for practice or research, in the urban areas he finds himself generally without much employment and considers it lucky if he secures a bare pittance by allowing himself to be exploited by the authorities in charge of the hospital administration in India. The short-sighted policy we have followed so far of paying as little as possible to men who are called upon to do serious and responsible work must give place to one whereby young obstetricians and gynæcologists in our hospitals should be paid a decent allowance to keep them above want and to allow them to bring a contented, fresh and vigorous mind to bear upon their work. More than this we should try and make them more self-conscious and self-reliant by giving them positions of trust and responsibility instead of, as now, merely making them do the inferior work and monopolising all responsible work in our own hands.

To successfully overcome the difficulties arising out of this overcrowding in cities it is moreover necessary that the State should, for the present, subsidise the specialist practising in the district so as to attract more and more men in the mofussil. There are various ways of doing this. One practical suggestion I can make is to impose medical cess in the rural areas like, for example, the road cess, education cess etc., and distribute it amongst chosen medical practitioners and midwives in the districts. Where the individual alone cannot pay, the State must, if it is to discharge its primary obligation of maintaining the health of the nation, devise means to attract the right type of men to the district and sub-divisional towns. What is needed at present is a joint endeavour by the State and medical men in this behalf. It is idle to hope that an ambitious young gynæcologist who has spent time, labour and money over training himself for his vocation will stick to the mofussil unless is is assured of gains commensurate with his skill and equipments and of opportunities for higher works.

A very important problem, again, which the war has forced upon us and which is looming very large before our eyes is the replacement of foreign drugs by the manufacture of indegenous drugs etc. I am glad to notice that organised activities by medical men have, of late, been set up towards exploiting the resources at our disposal for such manufacture. India is a vast store-house for drugs. Ancient India has long known the value of its own drugs and medicines. We have hitherto been guilty of criminal neglect of our own resources and of slavish dependence upon foreign drugs and medicines. The time has come when through joint endeavour and planned scientific research we can produce drugs and medicines suited to our requirements. No one will dispute the immense possibilities in this field and the great gains we can make by suitable and sustained work here. I do not see why we shall not succeed in mobilising Indian capital towards this end and why the Indian Research Fund and similar institutions should not extend a helping hand in this important work. The benefit to the suffering humanity, both economic and otherwise, out of the manufacture and use of Indian drugs and medicines, cannot be over-emphasised.

Ladies and Gentlemen, I have given you a bare outline of some of the many problems with which we as obstetricians and gynæcologists are vitally concerned—problems which cry for solution if we are to do any good to our motherland. It is clear to my mind that none of us, however well-meaning we may be, can achieve anything great unless we work unitedly. Jointly we have to devise plans and carry them out with meticulous care. To this end, it is necessary that we should start as many obstetric societies in the country as possible. The most important work in the field of maternity and child welfare is, of course, the antenatal and postnatal care of the mother and child. All civilised countries are daily making

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EPIDEMIC DROPSY

Epidemics of this dropsy have been occurring amongst the rice-eating population of this country since 1877. In Calcutta, it was noticed during the years 1877-78-79-80 when Colonel Kenneth McLeod named it "Epidemic Dropsy." Further outbreaks were reported in 1901, 1907, 1926, 1934 and 1937. Following Calcutta, the disease was reported in various places, both in and outside India *e.g.*, Mauritus, Benares and Assam. During every epidemic it has claimed a large number of victims. The chief danger of this disease lies in its effect on the heart and the residual effects in different organs.

Previously much controversy existed regarding the exact nature of the heart lesion till the pathology of the disease was worked out by Acton and Chopra (1927)[1], Shanks and De (1931)[2] and others[3,4,5]. It was found that the heart was enlarged, the right ventricle being more affected than the left. The ventricular cavities were dilated and the muscular walls were thinner. No inflammatory exudate was found on the pericardium but the sac sometimes contained an excess of fluid. Microscopically, there was an increase in the number of dilated and engorged capillaries. The muscles, however, never showed any damage and in extreme cases they were separated from each other by the dilated capillaries. Such a picture is never met with in any other disease. This separation naturally interferes with the co-ordinated contraction of the muscle fibres and this diminishes the functional activity. The dilated and engorged capillaries in the heart are a part of the generalised "capillary crisis" most marked in the legs.

The clinical picture has been critically studied by Chopra *et al*[6,7]. It has been found that cardiac involvement is almost a rule but the severity is not uniform. Various forms are seen in an epidemic. From a transient swelling of the legs to acute or subacute forms are met with. In an acute form it resembles acute congestive cardiac failure with slight cyanosis, jugular distension, and general systemic venous congestion. Generally, in subacute cases patients recover whereas in fulminating cases death may occur in 4 to 7 days. The cardiac symptoms are associated with low blood pressure and tachycardia. In the epidemic at Purulia (Chopra *et al*[8]), 54·5 per cent of the cases had palpitation and enlargement of the heart (in varying degrees) was found in 21 per cent. Ghosh[9] in a series of 154 cases treated in the outpatients department found palpitation in 72 per cent cases and dyspnœa in 50 per cent. Electrocardiographic records show tachycardia in about 90 per cent with regular rhythm. Sinus arrhythmia is found in some cases. Although clinically and in postmortem studies the heart shows enlargement it is peculiar that only in 3 cases some degree of ventricular preponderence was recorded (Chopra *et al*[10]). By teleroentgenography (Chopra *et al*[11]), measurements of the left ventricle and the right auricle were found to be increased in 70 per cent. There is thus some lack of consistency between the electrocardiographic and other findings. It must be admitted that electrocardiography and teleroentgenographic measurements were not done in the same group of cases and also the degree of cardiac involvement was perhaps not the same in the two groups. This may be the cause of difference in the results. Further electrocardiographic investigations are needed to clear up this point. The teleroentgenographic records of epidemic dropsy are in contrast to the measurements in beriberi, recorded by Raman published elsewhere in this issue. The enlargement is general and the effect of vitamin B_1 administration is marked.

Apart from the heart involvement during the course of the disease, epidemic dropsy leaves behind a miserable sequel, especially in those cases where the heart and capillaries are badly affected. These patients become chronic heart invalids. They get attacks of palpitation, swelling of legs and a less capacity for work during every epidemic year or otherwise. This is hard to explain since we possess no data that an attack leaves behind any permanent damage of the myocardium. In some cases palpitation is the only sequel while others present the symptoms of effort syndrome. Though genuine cases of relapse are not uncommon, the intense propaganda by the doctors, of the heart involvement in epidemic dropsy since 1910, might be responsible for labelling genuine cases of effort syndrome as epidemic dropsy heart.

Treatment is symptomatic. Often remarkable improvement occurs with a change to a dry climate. But this is for those lucky few who can afford it. No specific treatment is known since the cause is still unknown. It has been proved that the

[1] ACTON, H. W. AND CHOPRA R. N. (1927)—*Indian M. Gaz.* 62:359.

[2] SHANKS, G. AND DE, M. N. (1931)—*Indian J. M. Research*, 19:466.

[3] DE, M. N. (1933)—*Calcutta M. J.* 27:229.

[4] DE, M. N. AND CHATTERJEE, K. D. (1935)—*Indian M. Gaz.*, 70:489.

[5] TRIBEDI, B. P. AND DE, M. N. (1940)—*Calcutta M. J.*, 37:209.

[6] CHOPRA, R. N. AND BASU, U. P. (1930)—*Indian M. Gaz.*, 65:546.

[7] CHOPRA, R. N. AND BOSE, S. C. (1933)—*Ibid.*, 68:605.

[8] CHOPRA, R. N. AND CHAUDHURI, R. N. (1935)—*Ibid.*, 70:482.

[9] GHOSH, J. (1941)—*Calcutta M. J.*, 38:118.

[10] CHOPRA, R. N., CHAUDHURI, R. N. AND DE, N. (1937)—*Indian M. Gaz.*, 72:1.

[11] CHOPRA, R. N., CHAUDHURI, R. N. AND SEN GUPTA, P. C. (1937)—*Ibid.*, 72:395.

disease is not due to the deficiency of vitamin B_1. Vitamin B_1, given therapeutically or as a preventive measure, has no effect on the disease. So "many theories regarding the disease have been formulated, have lived their day and became history and have been revived again"[12]. In 1909, Rai Bahadur Hari Nath Ghose thought it to be due to an infection of the rice by a fungus. Later on, the spore-forming proteolytic bacillus of the bacillus vulgatus group infecting the rice was attributed as the cause of this toxic syndrome. For some years this theory held the field. Dr. Satya Saran Mitra came to the conclusion that the disease was due to adulterated mustard oil almost simultaneously as the rice infection theory. But this was not accepted at that time. During recent years, the adulterated mustard oil theory has been revived by Lal *et al*[13]. They could produce the disease with the adulterated mustard oil on human volunteers. The offending agent is the accidental mixture of Argemone mexicana seed with the black variety of mustard seed, both of which bear a superficial resemblance[14]. It is supposed that a 5 per cent mixture of this argemone oil in mustard oil ingested in normal doses is sufficient to produce the disease in 15 days. In this connection it is interesting to note that in the epidemic at Cawnpore in 1937 (Banerji[15]), the disease broke out amongst non-rice-eaters which included Marwaris, Anglo-Indians, and Europeans. Some of the sufferers used "til" oil instead of mustard oil. On the other hand, in the epidemic in Sierra Leone reported by Burnett[16], the disease occurred in a rice-eating population who used palm oil. However, both schools agree that a toxic body is responsible for the disease. While investigating the evidence of the toxic body in the plasma, on cell growth in tissue cultures, it was seen that some of the degenerated cells showed cytoplasmic granules and attempts were made to propagate a possible virus. This work, unfortunately, has not been further followed up. Others have suggested that "fundamental cause of epidemic dropsy is a dietary deficiency, which permits of an otherwise inocuous infection and the production of toxins with all the attendant signs, symptoms and pathological changes".[17] Dietary deficiency has never been so serious as at present, when famine condition is prevailing in the eastern provinces of India. With the scarcity there has been hoarding or storage of rice in some quarters. Mustard oil as is now available could not be more adulterated. Nobody has yet taken the trouble to investigate the amount of argemone oil contamination. Taking into consideration the above factors and the onset of rainy season, are we heading towards another epidemic with its mortality and morbidity to add to the already appalling figures?

It looks that after half a century through the ravages of this fell disease with their crippling effect on the heart, our knowledge regarding the disease is still foggy.

[12] Editorial (1935)—*Indian M. Gaz.*, 70:511.

[13] Lal, R. B. and Roy, S. C. (1937)—*Brit. M. J.*, 1:1110.

[14] Lal, R. B. (1938)—*Calcutta M. J.*, 35:169.

[15] Banerji, N. D. (1937)—*Indian M. Gaz.*, 72:675.

[16] Burnett, W. A. (1933)—*Ibid.*, 68:651.

[17] Editorial (1937)—*Ibid.*, 72:618.

PARENTCRAFT

Mukherjee[1] in his Presidential Address in the Section of Obstetrics and Gynæcology of the All-India Medical Conference, Patna, has urged that more education on motherhood, pregnancy, antenatal care, infant feeding and other kindred subjects should be included in the curriculum for girls. This is a subject which is engaging the attention of all people who are trying to better the lot of the mothers and children. We had the occasion to remark on the appalling ignorance in sex, confinement and the care of children in our country and time and often we have urged both the Government and the Public to deal with the matter in a straight-forward and practical manner so that the mistakes of our forefathers are not repeated in our generation or the generations to come. But, unfortunately, our appeals have so far fallen on barren grounds and very little advancement has been made in this direction in our country. The traditional taboo on sex has been kept up not only in India but throughout the whole world, despite the frantic appeals of Freud and his followers. Hence a statement to this effect from a senior practitioner is not only interesting but important. This definitely shows that a change in the outlook has taken place and that time has come when a further fillip should be given to the subject. This is also true of other countries of the World.

National Association of Maternity and Child Welfare Centres and for the Prevention of Infant Mortality organised a conference on Mothercraft teaching and Education for Parenthood in last October in London, where many interesting and important points were discussed. It was felt that the exclusion of mothercraft from the curriculum was definitely a short-sighted policy on the part of the State. And it was opined[2] that, "no girl should become a mother without having had some practical instruction about motherhood." Further, "A girl should have practical instructions in the details of how to manage the new-born infant, and also instruction about her own body and how to manage it." This conference after much deliberation, passed the following resolution:

"That, in the opinion of the delegates attending this conference, there is an urgent national need for the teaching of parentcraft to every potential parent. It is also their opinion that this teaching should be started during the year of compulsory education which should be extended forthwith."[3] These are very practical and important points which should attract the attention of all well-meaning educationists. Many a marriage had been made unhappy on the rock of the ignorance of sex. Very few girls are prepared for menstruation, very little is being done to impart the knowledge that it is natural and there is nothing shameful in it; very little has been done in the shape of instructing the prospective brides about sex, sex-life, marital relations, pregnancy, parturition and infant feeding and care. All these had so long been left to chance and probably to God with infinite misery, death and invalidism with consequent backwardness of national life. Now when the voice of the people

[1] Mukherjee, B. D. (1943)—*J. Indian M. A.*, 12:321.

[2] Nicoll, A. (1943)—*Mother and Child*. 13:179.

[3] *Mother and Child* (1943), 13:200.

have turned to this question, even in our country, we should try to give effect to the practical teaching of parentcraft to both boys and girls, so that they can launch into the marital adventure with full knowledge of what can be expected out of it and how one can enjoy it to the full to the betterment of the whole society.

Ancient Hindus understood the problem so well that they could produce so beautiful monographs on the subject as Batsayana's Kamasutra. They have formulated that one must not marry while he is a student and they came to the conclusion that the art of marriage cannot be known unless specially learnt either by discussions with those who know about this art or from meetings of the citizens, specially called for this purpose. "Girls", Batsayana[4] has said, "should study sexology in her parents' house before puberty." And he included as many as 64 sciences in his curricula for happy life. With the modern life, further complications have come into our lives and a thorough understanding of the responsibility and inner meaning of sex life and parentcraft has become a necessity in India as anywhere in the world to-day.

Recent investigations have brought out further facts to illumine the subject. These are biological factors of parenthood. Burns[5] of the Durham University in a thorough investigation continuing for about seven years has come to very important conclusions. She has shown that the age of the mother has got a special bearing on infant mortality, and a high death rate is found amongst infants born of mothers below 20 and over 30. The second factor contributing to the problem is suitable spacing of the family. In our country these factors will further be re-inforced by undernourishment, over-work, lack of rest, superstition, lack of knowledge and malhandling during labour.

Burns has also asked us to face the question of infant welfare from the eugenics point of view. We must ask whether we are saving people who will be able to carry on in the life or we are, by saving the unwanted infants, allowing the misfits to crowd the face of the world. It is a common knowledge that to-day breeding has almost been left to the poor while the rich are resorting to various contraceptive methods with the consequent limiting of their family, so the time has come to look into the matter with commonsense and broader outlook.

[4] BATSAYANA—Kamasutram. Chap. 3, verse 2.

[5] BURNS, C. M. (1942)—Infant and Maternal Mortality in Relation to Size of a Family and Rapidity of Breeding. A Study in Human Responsibility T. M. Carr, New Castle-upon-Tyne.

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P. B. MUKERJI,
Hony. General Secretary, I.M.A.

CURRENT COMMENTS

MALARIA PROBLEM

In the columns of this Journal, the problem of malaria has been discussed many a time. But the problem, though an old and permanent-type of friend of ours, very little progress has been made by the Public Health Authorities towards its control. With the advent of war, and speedy transport of troops from place to place, in malarious districts, it has attained the dignity of a world problem. Naturally, the whole world has become interested in the problem. We have collected here a few of the discussions touching the control of quinine and treatment of malaria.—EDITOR, *J. Indian M. A.*

QUININE CONTROL IN BENGAL

Bengal Government has decided to control Quinine consumption in the Province according to the following scheme:—

1. Quinine will be supplied to the District hospitals according to the endemicity amongst the local population.

2. This will be decided in consultation with the District Magistrate, Civil Surgeon and Health Officer of the District. Director of Public Health will act as the sole head for distribution of Quinine.

3. The controlling authorities in each District will first supply to the Charitable Dispensaries for free distribution amongst the poor, the rest will be given to the pharmacists and (in village) to the practising physicians.

4. The controlling authority will fix the quota to be allowed to each vendor and issue permits for the same.

5. Quinine will be sold on prescription of registered medical practitioners. Those physicians who will be allowed to sell Quinine shall have to first prescribe and then sell Quinine. They shall have to keep records of each sale. Not more than 90 grs. of Quinine will be allowed for each patient and no one will be allowed to try more than 45 grs. at a time.

6. Price of Government Quinine will be as follows:—

Powder gr. 5, one anna. Tablet gr. 5, one anna; 9 such, seven annas. Mixture gr. 5, one anna; Outside Calcutta 9 marks, eleven annas.

7. Each dealer shall have to show: (*a*) Government fixed rate for Quinine, (*b*) Quinine in stock at the beginning of the day.

8. This scheme has been started from this year.

GOVERNMENT QUININE RESTRICTIONS (IN AMERICA)

The War Production Board has forbidden the sale, transfer, delivery or consumption of quinine and the other cinchona alkaloids and salts for purposes other than antimalarial treatment. Nevertheless, pharmacists are reporting to the Board that physicians still prescribe cinchona derivatives for such conditions as the common cold and amenorrhœa. They are supposed to indicate when they prescribe quinine that it is "for antimalarial use." We must all bear in mind that the cinchona derivatives are under strict control; that they are intended solely for the treatment of malaria; that they can no longer be used for such conditions as the common cold; that many pharmacists have voluntarily contributed their surplus standing stocks of antimalarials to the Government through the "National Quinine Pool," that totaquine, now official in the U. S. P. XII, is satisfactory substitute, grain for grain, for quinine sulfate; and that full medical approval has been given to the use of kuinacrine, U. S. P. XII and pamaquine, quinine and urea hydrochloride and urethane may still be used.—Editorial, *Medical Times*, 1943:71:1.

TREATMENT WITH MEPACRINE

The curative dose of mepacrine—i.e., the dose used in treatment of an attack of malaria (we refer only to the adult dosage)—is 0·3 gramme taken in three spaced doses (three 0·1 gramme tablets) a day, preferably after food (or with milk), as there is slight tendency to minor gastrointestinal symptoms. Usually after a mild purgative this dosage is begun early and continued for seven days. If the fever has not subsided by the end of this period it is to be suspected that some other condition than malaria is present. The reasons why administration is not continued in any one course for a longer period than seven days are

(1) that it is not necessary, and

(2) that mepacrine has a rather marked cumulative effect. There is no objection, however, to a repetition of such a course after an interval, say, of a week's time.

This simple procedure constitutes the essentials of the treatment of a malaria attack—namely, the temporary destruction of the parasites. It is very doubtful whether with either mepacrine or quinine more prolonged treatment at this time will effect any more complete destruction and one must trust ordinarily to treating as a further attack any relapse which may subsequently develop, though something may be done in the way of after-treatment to reduce the chances of this. One consideration in treatment is very important and requires constant vigilance and prompt action; this is when dealing with falciparum infection, to anticipate the onset of coma or other pernicious manifestation indicating serious danger of death. Falciparum infection, or so called malignant tertian, is the common malarial infection contracted by Europeans in the tropics. In spite of the name attacks are not necessarily severe, but the parasite has the property not possessed by any other malarial parasite of giving rise, often rather suddenly or insidiously, to pernicious manifestations the normal end of which, unless promptly countered, would be death. The indication in such an event is to get the specific drug at all costs at once into the circulation, and it is usually taught that an immediate injection should be given intravenously, this being the quickest known route of administration. Intramuscular injection is also recommended by some authorities, but in the case of quinine there is a divergence of opinion. With mepacrine the local necrotic effects produced by the quinine salts usually used appear to be absent, and intramuscular injection, which need not be into the gluteal region, has been widely employed and apparently without objectionable effects. If intravenous injection is difficult, intramuscular administration of mepacrine may, therefore, be substituted, but intravenous injection is perhaps more rational and better practice. The precaution should be taken to inject slowly.

The *usual prophylactic dose of mepacrine* is 0·2 gramme (to 0·1 g. tablet) a day, given on two successive days each week. This is roughly the equivalent of the usual 5 grains of quinine daily. Mepacrine in certain amount produces a yellow coloration of the skin. This is quite harmless and merely due to the colour of the dye. It is in no way related to jaundice, nor is it evidence of constitutional disturbances.—Editorial, *Brit. M. J.* 1943:1:75.

Drugs Used in Treatment of Malaria

With regard to drugs in the treatment of malaria, before the last war we were told—from the teaching of Prof. Dixon in Cambridge—that the most specific of all drugs was quinine. Yet as this war progressed we began to disbelieve more and more in its specificity, and had to modify our fundamental ideas. We believe now that quinine is an extraordinarily valuable drug but is definitely selective upon certain stages of the malaria parasite. The species of the parasite on which it acts most quickly is the benign tertian, but we cannot entirely banish it from the human body by one or two or even three doses of quinine. The plasmodium has a way to side-tracking and slipping away into the interstices of the bone marrow and the spleen, ready to emerge again at the next opportunity. This selective action of drugs was brought out strikingly when plasmoquine (pamaquin) was discovered in 1925; it is a by-product of methylene blue, which was employed by Manson in China as a febrifuge many years ago.

When the blood is full of subtertian crescents and plasmoquine is given in sufficient doses this stage of the parasite, which is not amenable to quinine, rapidly disintegrates. The parasites burst at various points: the chromatin degenerates, and finally they vanish from the blood stream.

Atebrin (or mepacrine), an acridine compound, is another dye derived from methylene blue. It does not affect this crescent stage at all and has no effect upon exflagellation, but causes the pigment to collect in the centre of the parasite.

Diagnosis—it is the differential diagnosis of malaria which is so important. Malaria usually causes enlargement of the spleen, but whether you can tell that this is so or not depends on your skill in palpation. In subtertian malaria the spleen becomes rounded off rather like a tennis-ball, but it is only after repeated malarial attacks that the classical hard edge can be detected. It is, therefore, not safe to say that a man has not got malaria because the spleen cannot be palpated. There are various ways of performing palpation, and new-comers have to learn the correct technique and the question arises; when is a spleen not a spleen? The answer is: When it is a kidney! An enlarged left kidney is sometimes mistaken for an enlarged spleen. There is splenic pain, which may be significant.

In the diagnosis of malaria the great point to remember is that in a malarious country it may simulate almost any other disease. Subtertian malaria is the greatest mimic. Quite commonly it produces cerebral symptoms—cerebral malaria, due to the congregation of parasites in the brain, sometimes leading to coma and death. Very often in the near East the worst forms of malaria are found in the winter-time. Mosquitoes once infected remain so, and in winter tend to congregate in buildings and nooks where there is warmth and then descend upon any one they can find. These dire cerebral forms may crop up during the winter months and may be mistaken for other forms of cerebral disease. In the summer the patient may go down with hyperpyrexia, and so the case may be mistaken for heat-stroke.

I would specially warn against confusing alcoholism with malaria. In a malarious country a man may be accused of being drunk when he is suffering from malaria.

Mania is another thing which may be misleading: the patient may be suffering from subtertian malaria delirium; and, on seeing a sudden epileptic fit, do not jump to the conclusion that it is true epilepsy for it may be malaria simulating that condition. Then there are attacks simulating cerebrospinal meningitis with pain and stiffness at the back of the neck. There is an appendicular form of malaria that is due to sporulating forms of the parasite which select that particular part of the intestinal canal in which to congregate. When the patient's temperature rises with rigor to 104° F. as a sequel to an abdominal operation, you will want to ask whether malaria parasites were not in the blood before the patient was subjected to operation or whether the operation actually incited the rigor. Cholecystitis is also a dangerous primary diagnosis. Why should an epidemic of cholecystitis in healthy young men suddenly arise? The explanation lies in the hepatic congestion in subtertian malaria, with an acutely distended gall-bladder.

Then there is malarial dysentery or colitis. Malaria is frequently accompanied by diarrhœa; in fact, often there may be diarrhœa and nothing else to guide one. It is very common to see patients from West Africa who have suffered from diarrhœa on the voyage and, owing to this, doctors have prohibited them from taking quinine. I have seen such cases develop cerebral malaria and fatal coma the moment the boat has docked.

In Egypt subtertian parasites have been demonstrated in scrapings made from the bowel. "Malarial dysentery" with blood and mucus in the stools simulates acute bacillary dysentery. It is possible to get such an acute diarrhœa with malaria that it resembles cholera. There were several cholera scares in the last war, but they were due to a choleraic form of subtertian malaria. Then we may meet the icteric forms; but malarial icterus is not a really true obstructive jaundice. In subtertian malaria it is almost diagnostic to get urobilinuria with sherry-coloured urine, and this may sometimes be taken as an index of infection, even in the absence of subtertian parasites in the blood. One can meet with forms resembling acute pancreatitis in which the pancreas is affected; others resemble intestinal obstruction, with profuse vomiting. One may even see cardiac forms with generalised oedema, which were frequently encounterd in the last war, or again pulmonary forms associated with bronchitis or pleurisy. Further, there may be some resembling acute nephritis with blood and cells in the urine; or a cutaneous rash with multiple hæmorrhages in the skin, somewhat resembling typhus or measles.

Blackwater Fever—Finally, there is the most dramatic sequel—blackwater fever, solely associated with subterian malaria. It probably represents an anaphylactic phenomenon due to hypersensitivity to toxins of this parasite. The prevention of blackwater fever is closely linked up with the

prevention of malaria so that if a man does not get infected with the subtertian parasite he will not develop blackwater. If a laboratory service is maintained by which in all cases the blood is examined for malaria the moment the patients fall ill, then the onset of blackwater fever will be prevented. A patient is exposed to the danger of blackwater fever once he has had acute malaria—usually up to nine months. It is apparently a case of supersaturation with this parasite, so that blackwater seldom results after a primary infection. It occurs mostly in the old residents of the tropics who, on returning to this country, have been exposed to cold and wet or some shock which causes a sudden multiplication of parasites. Then the red blood corpuscles break down and become hæmolysed, and the victim may die from blocking of the kidneys with products of their disintegration. If, therefore, this process can be prevented from taking place, then blackwater fever will not ensue. Treatment depends upon the alkalinization of the blood and upon prompt blood transfusions.

Prevention and Treatment—How are we going to prevent malaria in the field? Quinine in large doses is a poison; it may produce amblyopia in some people, and they may become blind. Quinine in enormous doses, as a means of preventing a malarial attack, is a broken reed. It has been shown that one can inject quinine together with malaria parasites into the blood of the recipient and yet produce malaria. On the west coast of Africa most officials cling to the belief that 5 grains of quinine will prevent malaria and blackwater fever. It probably modifies the acuteness of the malarial attacks and thereby the proclivity to blackwater fever, but when you give soldiers who are operating in open country 5 grains of quinine a day it has little visible effect in reducing the incidence of malaria. *For prevention on a large scale the chief line of defence is the mosquito net. The next best method is to kill the mosquitoes.* With regard to drug treatment of the actual attacks, however, quinine still remains the standard. It is most effective in benign tertian malaria and it has also a great effect upon subtertian. But it is necessary to remember that quinine given in big doses to a patient with a large number of subtertain malaria parasites is likely to precipitate blackwater fever by the sudden dissolution of the parasites, and the patient may die within twenty-four hours. For benign tertian malaria quinine should be given in dose of 10 grains three times a day and the bowels should be freely kept open. One must go on giving even the quinine in decreasing doses for about six weeks, though even this continuous therapy will not extirpate the infection from the body entirely; but to inject quinine intramuscularly to everybody indiscriminately is to ask for trouble, as abscesses and indurations are apt to form in a certain number. At the same time I never hesitate to give quinine intramuscularly when it is necessary as a remedial measure in severe infection. The results are magical if it is given in the right way and injected deeply into the gluteal muscles, *but it should never be injected below the folds of the buttocks.*

You cannot treat malaria scientifically unless you control treatment by frequent blood examination and can recognise the changes in the parasites when acted on by quinine or other drugs. Intravenous quinine is reserved for the most severe forms of subtertian malaria. From 5 to 7 grains dissolved in 5 c. cm of distilled water in quite enough.

With regard to plasmoquine, remember also that this is poisonous. When it was first introduced we gave it in too big doses, and were thereby producing poisonous effects. It is now recognized that small amounts of plasmoquine in tablet form known as plasmoquine compound are beneficial, each tablet containing 1/6 grain, with 2 or 3 grains of quinine. This, given as a regular course, has been found by the Army in India to be the best method of treating benign tertian malaria and of preventing further relapses. In such cases a start is made with the quinine and continued with plasmoquine compound, the dose of which is two tablets twice a day for a week, amounting to four courses of seven days, with a break of four days, between each. This means that about 2/3 grain of plasmoquine is given a day in addition to 20 grains of quinine. With this method the relapse rate in benign tertian malaria has fallen from 40% to something like 8% in the Army in India.

Plasmoquine and atebrin go badly together but atebrin and quinine work well in combination. The dose of atebrin is 1/10 gramme three times daily for seven days. It is a yellow tablet which is given by the mouth; but there is also a soluble form which can be injected. It is active especially in the treatment of subtertian malaria, and I have seen only one case in whch blackwater fever supervened after atebrin. So it is useful in this direction. I do not believe, however, that it is nearly as effective in treating benign tertian malaria as the method I have outlined. Certain disadvantages of atebrin have been pointed out, and it is well to warn people that they may acquire a somewhat yellowish tinge. I have never seen any body go "off his head" when taking atebrin, but acute mania in native races has been described after taking it in therapeutic doses, though this certainly does not seem to be common in Europeans.—Sir P. H. Manson-Bahr in *Brit. M. J.* 1942:2:489.

XX ALL INDIA MEDICAL CONFERENCE, 1943

The XX Session of the All India Medical Conference will be held at Ahmedabad in X'mas Week 1943.

CURRENT MEDICAL LITERATURE

MEDICINE

Wasting Disease and Lesions of the Nervous System

Hsii (*Arch. Neurol. Psychiat.*, 1942:48:271, *Ref. Brit. M. J.*, 1943:1:195) carried out a very thorough histological examination of the central nervous system in 13 subjects who had died from wasting disease associated with dysentery and tuberculosis. In every case distinct lesions of the intestine were demonstrated. The changes in the nervous system were remarkably uniform in nature, including primary irritation of the Betz cells and degeneration of the dorsal spinal ganglion, posterior columns, posterior nerve roots and peripheral nerves. In beriberi peripheral nerves are primarily affected while in pellagra the brain appears to be the earliest site of disturbance. The symptoms of vitamin B deficiency were recorded in only one case but it is possible that these manifestations had been overlooked in the others. The author indeed, after an exhaustive search of the literature and analysis of his own findings, has come to the conclusion that the combination of pathological changes points to the deficiency of the vitamin B complex. Whether or not this is the case, it raises the interesting problem of the organic basis for the neurological abnormality that arises in the course of general diseases. The association between disorders of the alimentary tract and diseases of the nervous system has long been suspected. The author stresses on the importance of an exhaustive neuropsychiatric examination in all those showing disturbances of the gastrointestinal symptoms. It must be left to experimental work to decide whether disease of the alimentary tract acts directly on the central nervous system presumably by cutting down some hormonal effect or indirectly by impairing the absorption or utilization of one of the vitamins. Meanwhile it might be wise again to emphasize the importance of adequate vitamin intake for all patients with digestive disturbances.

Peptic Ulcer in the Aged: A Clinical and Postmortem Study

Mayer and Saphir (*Am. J. Digest. Dis.*, 1943:10:28) in reporting on the study of 16 cases of acute peptic ulcer in patients over 60, point out that while it is generally accepted that chronic peptic ulcer occurs in the aged and that such patients die from the complications of hæmorrhage and perforations of a chronic ulcer, the study directs attention to the occurrence of acute peptic ulcer in the aged. These acute ulcers have been observed following acute febrile disease, operation for gall stones, resection for carcinoma of the stomach, carcinoma of the prostate, prostatic resection, carcinoma of the lungs, congestive heart failure and coronary disease. Usually the occurrence of these ulcers is most often asymptomatic with reference to the gastrointestinal tract; the abdomen is not rigid even in cases with acute perforation. The authors doubt that the degree of acidity or accumulation thereof plays an important role in the genesis of ulcer in the aged. It seems more likely that as a result of increasing sclerosis of the larger and smallest arteries, local necrosis occurs which favours the development of the ulcer. During operation or postoperatively a fall in the arterial blood pressure may play a role in the causation of these ulcers. Vitamin C is diminished in the aged and this lack may be a predisposing factor not only in the genesis of the ulcer but also in the rapidity of development of the ulcer and its progression to perforation. Macroscopically acute ulcers in the aged simulate those seen in the younger age group. Histologically there are typical arteriosclerotic changes with marked intimal thickening and reduction of the lumen with the only difference that these changes are definitely more severe than those usually encountered in the earlier age group.

Chest Pains in Patients with Mitral Stenosis

Burgess and Ellis (*New England J. Med.*, 1942:226:937) in reporting 6 cases illustrating the different types of chest pain that may occur with mitral stenosis observe that four types of pain occur, *viz.*, a cardiac neurosis, the pain of acute rheumatic carditis, angina pectoris and so-called hypercyanotic angina, cardiac neurosis; the pain is of a very variable nature usually felt in the apical region but sometimes over the precordium, not brought on by exertion and persists for hours and even days. Acute rheumatic carditis during acute recurrences of rheumatic fever may develop chest pain which will be relieved when the acute symptoms subside. True angina pectoris is rare in mitral stenosis but may occur. Hypercyanotic angina usually occurs in advanced cases of mitral stenosis with cardiac pain very similar to that of an infarction but not accompanied by other typical symptoms, *viz.*, fever, leucocytosis and electrocardiographic changes; the accompanying cyanosis is often extreme and dyspnœa and orthopnœa severe but the pulmonary congestion may be slight; in 2 out of 5 cases reported, autopsy showed no evidence of myocardial infractions.

Dangers of Sulphathiozole

Lederer and Rosenblatt (*J.A.M.A.*, 1942, 119, Ref. *Med. World*, 1943:57:529) call attention to the dangers of this remedy and describes 4 cases in each of which the drug was given uninterruptedly. During the treatment there occurred a sudden onset of rigors and fever, sometimes associated with a rash, conjunctival injection or myalgia and arthralgia. These symptoms were believed to be due to infection and so the drug was continued in the original or even in increased doses. The fever, however, persisted, coma rapidly developed and in two cases anuria became a prominent feature. All of the four cases died. It is concluded that the indiscriminate and uncontrolled use of sulphathiazole is not without risk. The urine should be regularly examined during the treatment as the finding of crystals of the drug in the urine and the occurrence of hæmaturia are danger signals. In conclusion sulphathiazole should only be used when it is definitely indicated and ought not to be continued longer than is absolutely necessary.

Nervous Symptoms due to Sulphonamides

Little (*J.A.M.A.*, 1942:119, Ref. *Med. World* 1943:58:114) writes that sulphonamides may cause quite a number and variety of nervous phenomena which include aphasia, stammering, peripheral neuritis, blindneses, convulsions, transitory myopia, meningeal signs, agraphia, toxic psychosis, myelitis and encephalomyelitis. This group of drugs appears to be more toxic if ad-

ministered to patients already suffering from disease of the nervous system, for in such cases neurotoxic symptoms are likely to develop.

SURGERY

Resuscitation*

Organe (Proc. *Roy. Soc. Med.*, 1942:35:439) in a paper read at a recent meeting of the Section of Anæsthetics of the Royal Society of Medicine, observes that death on the operating table is due to heart failure, which may be primary or secondary.

Primary heart failure is due to cardiac standstill or to ventricular fibrillation. Cardiac standstill probably results from vagal inhibition excited reflexly from the respiratory tract or from a painful stimulus elsewhere. The heart may be healthy and will resume its normal rhythm on the application of a suitable stimulus. Overdose of anæsthetic, even of chloroform, produces respiratory failure before cardiac failure. Ventricular fibrillation is produced by the same stimulus of vagal inhibition in the presence of an abnormally irritable myocardium. This irritability may be produced by chloroform or cyclopropane and is greatly increased by adrenaline. Adrenaline alone, in very large doses, can produce ventricular fibrillation with other anæsthetics or in the unanæsthetised subject. Vagal inhibition of the normal pacemaker of the heart releases lower ventricular foci of irritability which are not under vagal control, with resultant multifocal ventricular tachycardia leading to ventricular fibrillation. Sudden heart failure under ether anæsthesia is probably never due to ventricular fibrillation except where large amounts of adrenaline have been used.

As adrenaline acts directly on the ventricles it seems likely, even under ether anæsthesia, that intraventricular injection of adrenaline may convert cardiac standstill into ventricular fibrillation.

Secondary heart failure is due to anoxia of the myocardium, and occurs earlier with ethyl chloride or chloroform owing to the toxic effect of the anæsthetic.

Treatment may be preventive or restorative. Preventive treatment, apart from the avoidance of grosser errors, is of little effect in primary heart failure. Restorative measures are likely to be ineffective in secondary heart failure.

Preventive treatment in secondary heart failure. Maintenance of blood pressure: Where blood pressure fall is due to peripheral vasodilation, as in spinal anæsthesia, vasoconstrictors may be used (neosynephrine 0·05-0·1 c.c. or ephedrine 15-30 mg. intravenously). The systolic blood pressure should not be allowed to fall below 80 mg. Hg. Maintenance of blood volume lost from shock, hæmorrhage or transudation of plasma. A systolic blood pressure of 90 mg. Hg. or less due to shock or hæmorrhage requires correction. In an early case, intravenous normal saline may suffice. In most cases serum or plasma should be given. Whole blood is necessary after severe hæmorrhage or if there was anæmia before operation. Injection of so-called cardiac stimulants may be dangerous in a patient gravely ill from shock or hæmorrhage. Maintenance of oxygen supply. The supply of oxygen to the blood may be reduced by respiratory obstruction; by too little oxygen in the anæsthetic gases; by deficient pulmonary ventilation; by impaired gaseous exchange (after poison gas or due to pneumonia); by anæmia. The proportion of oxygen administered may be raised to 30%. If breathing is shallow, artificial respiration or respiratory stimulants should be given. (Carbon-dioxide 5%, picrotoxin 3 mg., cardiazol ½-1 c.c., coramine 1-2 c.c., or alphalobeline 5 mg., intravenously). If respiratory exchange is impaired, the oxygen percentage should be as high as possible. Withdrawal of the anæsthetic (or neutralisation of barbiturates or avertin by analeptics) is essential.

Preventive treatment of sudden heart failure. Pre-operative atropine or scopolamine to inhibit the vagus; avoidance of chloroform. avoidance of excitement during induction of anæsthesia and of premature operative stimuli; great caution in use of adrenaline, which is absolutely contraindicated with chloroform or cyclopropane.

Restorative treatment of primary heart failure. (1) Clear the airway and establish artificial respiration with oxygen. (2) Mechanical stimuli to the heart. (*a*) Auricular puncture with any needle of 3½ inches (9 cm.) or more in length, piercing the chest wall in the third right intercostal space ½ inch (1·25 cm.) from the border of the sternum and passing inwards and towards the midline. Irritability to mechanical stimuli is soon lost from anoxia and auricular puncture is less dangerous than delay. (*b*) If auricular puncture is not immediately successful, cardiac massage must be started through an upper abdominal wound. (*c*) If massage is not effective within four minutes of the onset of heart failure, an incision must be made in the diaphragm and an artificial circulation established by direct massage at a rate of forty compressions a minute. (3) Adrenaline. If a satisfactory artificial circulation is not established, or if spontaneous contractions have not appeared in ten minutes, inject 0·5 c.c. of adrenaline 1/1000 solution into the ventricle through the fourth left intercostal space 2 inches (5 cm.) from the border of th sternum. By this time, cardiac irritability is reduced and ventricular fibrillation is not likely to occur. (4) Procaine. If the surgeon attempts cardiac massage and finds ventricular fibrillation, inject into the cavity of the ventricle 10 c.cm. of 2% procaine.

If a satisfactory circulation is not established within four or five minutes, permanent damage to the brain is likely to occur, leading to death at a later period.

Chemical Changes in the Blood and the Occurrence of Uraemia following Head Injury*

Cumings (*J. Neurol, Psychiat.*, 1942:5:40) writes that as a result of investigations made upon victims of air-raids it has been established that uræmia occurs as a result of crush injuries of the limbs. The occurrence of uræmia as a complication of head injuries without damage to any other part of the body has not been recognised. The present author describes the results of investigations into the chemical changes in the blood of 25 patients, with head injury of varying severity and with no other lesion, who were examined 1 to 13 days after injury. The only abnormality found was a tendency for a raised inorganic phosphate in the plasma of patients examined some days after a severe injury. Seventeen further patients were examined with reference to renal function. Only cases with severe lesions producing unconsciousness of some duration were chosen, and in 10 of these a raised blood urea was found. Four of these—all young adults—developed uræmia, and of them three died. Autopsies were performed in two of the fatal cases. The

patients with uræmia were incontinent, and passed a normal volume of urine which was acid and contained a normal amount of urea. There was, however, a rather low chloride content. Chemical analysis was performed on the kidneys removed at autopsy, and the only abnormality found was a raised uric acid content in the renal medulla in one case. Histologically the only abnormality found was in the kidney and was limited to damage to the proximal part of the first convoluted tubule. One patient with uræmia improved on alkali treatment in the form of large doses of potassium citrate and sodium bicarbonate by mouth.

Treatment of Scalp Wounds in Air-Raid and other Casualties†

Botterell and Jefferson (*Brit. M. J.*, 1942:1:781) write that when scalp wounds are properly treated the injured are able to return to duty quickly and without loss of efficiency, but these wounds are sometimes regarded as unimportant and left to the care of inexperienced surgeons. Bad treatment consists in failure to observe deep penetration, but more often in failure to ensure primary healing (especially for subdural hæmatoma).

The authors strongly recommend that the shaving of the head should be done over a wide area, if not completely, before the patient comes into the operating room. This may be done with a safety-razor blade held in artery forceps, and common soap gives a better lather than the special surgical kinds. Because in air-raid casualties the wounds are full of brick-dust, plaster and cement powder, they are very difficult to clean. The authors recommend washing the wound and the surrounding scalp very freely with soap and water; 1% procaine solution with adrenalin (3 drops to 30 c.c. of anæsthetic solution) is then widely injected. When rendered insensitive, the wound is scrubbed with soap and water until it is entirely clean. In some cases ingrained dirt on the pericranium must be cut out, and sometimes a nail brush must be used. The effects of soap and scrubbing on the wound are not injurious because the wound is afterwards treated by careful debridement. The wound edges are freshened by excising a very narrow (2-3 mm.) strip of scalp. Large excisions must be avoided because of the inelasticity of the scalp. The wound is finally dusted with sulphathiozole or sulphanilamide powder and is then closed by a single layer of fine sutures. Neurological surgeons commonly use two layers of sutures, but it is enough, for a scalp wound with intact bone and dura, to use only one layer. Drainage is provided through a small stab wound about 2·0 cm. from the suture line. Dressings are firmly applied. The tube is removed after 24 hours and the sutures are taken out on the 4th day. It is a common mistake to leave the stitches in scalp wounds too long. It is quite possible for a surgeon to work on scalp wounds with only one assistant or even with none. If there is a shortage of staff it is better for assistants to concentrate on the preparation and after-care of the patients. Only those with cerebral contusion and mental confusion require a general as well as a local anaesthetic, and evipan or pentothal will best meet this requirement. Finally, the authors state that the teaching that head injury cases do not tolerate transportation needs revision. They believe that all the more serious cases should be transferred to one of the Special Head Centres now functioning in Britain as part of the Emergency Medical Service (Ministry of Health). The need for an excellent surface operation is emphasized, and the authors deprecate interference by general surgeons with deep wounds in the brain. The general surgeon should, in their opinion, be content to deal with the scalp and, if necessary, with the bone. The case can then be transferred to the care of the neuro-surgeon, who can deal with the more difficult parts later if the surface wound is clean and healing.

PEDIATRICS

Diphtheric Heart-Disorders in Children†

Neubauer (*Brit. M. J.*, 1942:2:91) from a study on a hundred cases of diphtheria with disorders of the heart in children observed from the onset to convalescence or fatal termination divides them into 3 groups: *The myocardial lesion.* This occurred in 38 cases. It may be diagnosed by bedside examination, but it has been found that a myocardial lesion may be detected by electrocardiography when the clinical signs are still doubtful or even absent. Myocardial damage may also still be found when the child appears clinically to be quite recovered. No child who has had a myocardial lesion should be discharged until the electrocardiogram has returned to normal. Failure to ascertain this may account for reports of slow recovery after diphtheria and for the tragic instances of children dying suddenly at home after discharge from hospital. *Abnormalities of rhythm.* Sinus-tachycardia occurs often in the febrile period of diphtheria and is of no special significance. Tachycardia which persists into later convalescence should be carefully investigated to exclude a myocardial lesion. Sinus-bradycardia is often found in the early convalescent stages. Gross bradycardia should always be carefully investigated. Sinus-arrhythmia is a common cause of cardiac irregularity in childhood and has no pathological significance. It should be distinguished from serious forms of irregularity as the activity of a healthy child may otherwise be quite unnecessarily restricted. Extrasystoles are not of serious import. *Interference with conductivity.* Partial auriculo-ventricular block which was found in 17 cases can only be definitely diagnosed by means of electrocardiography. Complete auriculo-ventricular block was found in 5 cases. The clinical as well as the electrocardiographic signs are very significant. Partial and complete bundle-branch block can be detected and definitely diagnosed only by means of electrocardiography. They occurred in 19 and 4 cases respectively. Auricular fibrillation does not occur only in severely damaged hearts with heart failure. It was also observed as a transitory phenomenon lasting for several hours or days without signs of myocardial involvement. The outlook in transitory auricular fibrillation is good, whereas the prognosis is very poor in cases in which congestion was present before the fibrillation started.

Sudden death within the first ten days occurs in severe cases of nasopharyngeal diphtheria due to the *gravis* strain of the organism. The fatal issue in these cases is usually brought about by one of three conditions: (*a*) gross myocarditis (*b*) complete auriculo-ventricular block, or (*c*) paroxysmal ventricular tachycardia.

Electrocardiographic investigation in support of careful clinical examination of the patient is desirable in the treatment of any but the mildest case of diphtheria. In general it is

possible to discharge a patient with a mild attack of diphtheria in 4 weeks. With an attack of moderate severity in 6—7 weeks, while those with a severe infection may have to be retained for 12 weeks or longer. Where evidence of myocardial lesion has been found the patient should not be discharged until the elctrocardiogram has returned to normal.

THE SURGICAL TREATMENT OF CONGENITAL DEFECTS†

MOWLEM (*Proc. Roy. Soc. Med.*, 1942:35:683) one of the leading colleagues of Sir Harold Gillies, the distinguished plastic surgeon, at a meeting of the Section for the study of Disease in Children of the Royal Society of Medicine read a paper on the subject the summary of which is given below:

The author points out that the plastic surgeon's chief contact with pediatrics is in the surgery of congenital defects. Not all require operations during early infancy but, as those who do are frequently in poor general condition, two main provisions are essential: the assistance of a skilled pediatrician both before and after operation, and a surgical team accustomed to meet and deal with the special surgical and anæsthetic conditions of early infancy.

Most of the rarer congenital defects can safely be left to the later years. *Hypospadias* and *epispadias* do not usually call for intervention until about the age of 6 years. In the former the reason for delay is the necessity of constructing the urethra to ensure both rectal and sexual potency. Usually only minor degrees of epispadias lend themselves to plastic repair, the alternative being ureteric transplantation. *Absence or atresia of the vagina* may also be detected in childhood but is not operated upon until puberty. *Syndactyly* and *polydactyly* are usually operated on between the second and fourth year, unless further delay is necessary for identification of the primary centres of ossification.

Hæmangioma.—The small raised cavernous type with capillary elements on the surface, which appear after birth is liable to spontaneous cure. The deep-seated truly cavernous type and the well-defined surface capillary type do not show such constant behaviours. The latter may improve, the former probably will not. The optimum treatment is either by radium or x-ray, but the initial radio-sensitivity of these growths decreases fairly rapidly after the first year. It may not therefore be justifiable to await spontaneous cure when the treatment is so simple.

Cleft lips and palates.—In the repair of these conditions the primary necessity is the provision of a normal speech mechanism. Repair after "palatal speech" has been established is not sufficient to restore normal enunciation and a long period of speech training will be required. In the author's opinion this disadvantage can be eliminated by operating sufficiently early to enable the child to develop spontaneously good speech and sufficiently late to be safe. His own practice is to operate at the earliest possible time. Although the first forty-eight hours is said to be the best, this is rarely possible. If this opportunity is missed it is unwise to give an anæsthetic until nutritional advancement is well established. The author's rule is to wait until the child weighs at least 10 lbs. (4·5 kg.). The repair of a complete cleft involving both lip and palate falls into two stages with an interval of three months between them, so that if it is hoped to complete surgical procedures before the end of the first year an early start is essential. With cleft of the lip only there is less urgency as there is no associated functional disability.

The disadvantages of such early intervention are chiefly increased technical difficulties. These can be overcome by the team which is accustomed to work on such small children but the team must be constant. In the author's experience the mortality rate for a large series of cases is considerably less than 1 per cent. In operations on isolated children in other centres without the use of the authors' usual team the mortality rate rose on one occasion to 60%.

The complications of early operation are chiefly respiratory. The great majority of small children show a reactionary temperature of as much as 100°F. (about 38°C.) on the day following operation. A few develop bronchitis, apparently due to nasal obstruction temporarily imposed by an adequate operation. Pneumonia is very rare. Both these complications can be reduced by confining the operations to the spring and summer months, necessitating postponement in some cases to a later period than would otherwise be justifiable. The risk of post-operative nasal infection which jeopardizes repair has been decreased by sulphanilamide which may be insufflated into the nose. Lastly, a rare complication, not often stressed, is hyperthermia pallida. The author has met with six instances, the first fatal. The child's temperature rises extremely high in the first few hours after operation. It is pale, has a respiration rate between 50 and 80 per minute, an uncountable pulse, and if untreated soon dies from exhaustion. In the fatal case no significant post-mortem findings were discovered. Though atropine is assumed to have a bearing on this condition, as much as 1/50 of a grain (about 1 mg.) has been given to one case without aggravating it. That it is due to so overloading the child in the theatre that its heat exchange mechanism is completely disorganized, is borne out by the lessened incidence since mackintoshes, pneumonia jackets and such impediments have been discarded. The treatment is to lower the temperature to about 97°F. (about 36°C.) by rectal washouts of ice water and to maintain it at that level for at least one hour. The heat-controlling centre apparently recovers and though there is usually a small rise of temperature on the next two or three days, the child's general condition remains good.

The difficulties encountered in operating upon small children are so great that success depends on close co-operation by the pædiatrician in preparing the child for operation and advising treatment during convalescence, and the surgical team equipped to deal with structures which are minute and an organism which is intolerant of prolonged surgical interference. Such a combination achieves good results and reduces the mortality rate to the minimum.

Abstracts marked with an asterisk (*) are received through courtesy of the United States Office of War Information, 293, Hornby Road, Bombay and those marked with a dagger (†) through courtesy of the British Empire Information Service, 3 Hanover Street, London, W. 1.—EDITOR—*J. Indian, M.A.*

NOTES AND NEWS

AN OMISSION

The portrait of the Late Sir Nilratan Sircar printed in the June 1943 issue of this Journal was from a block received through courtesy of the *Calcutta Municipal Gazette.*

PSYCHOLOGY FOR THE FIGHTING MAN

The subjects treated under the heading are: (1) why men fight; (2) zest; (3) how men defeat; and (4) fear: ally or traitor.

(1) There are twelve reasons listed for joining the armed forces. Whatever a man's motivation at the time of enlistment, however, when he is in actual combat he fights either out of loyalty to his unit, or because there is a leadership in all the uncertainty, or because there is literally nothing else to do. (2) Zest, a term chosen by the military for the commonly used term morale, is the weapon that makes a unit unbeatable. Leaders must know something of this variable and its dependence upon physical and mental fitness. In this connection, the cumulative effect of small disappointments and annoyances is shown as it operates to wear down the spirit of an organization with an incompetent commander. The friction which he generates in his junior officers percolates through the pyramid of the organization to harm even the buck privates at the base of the social structure. (3) The typical reactions to frustration and defeat are described. The number of times a man will attempt to reach a certain objective is governed in one direction by the importance of that particular success, and in the other direction by the effort and pain involved in trying. Sometimes in these attempts, a man must compromise, and occasionally he must accept defeat. Rationalization and hysteria are illustrated as harmful methods for accepting inevitable defeat. (4) Finally, fear is discussed. It is emphasized that every normal man is frightened in the face of danger. Even when terrified, however, a man who is well-trained will be able to perform mechanically. Six concrete facts that may be utilized for fighting fear are described: (1) Action dispels fear. (2) Physical contact with friends helps. (3) Roll calls help—they assure one that others are keeping track of him. (4) Men should be informed of the dangers they may expect, in order to eliminate surprise. (5) Control of action helps—panic-stricken men should be eliminated, and as much as possible every man must control the signs of his own fear. (6) Statistics showing the low frequency of casualties are reassuring. These points are particularly useful in times of prolonged peril. In tense moments, humor may be a lifesaver. And lastly, it must be remembered that religious belief may be a powerful antidote against fear. Foolhardiness is not bravery—those who know no fear are usually dangerous influences. Normal men show fear, but "none but the brave can afford to fear."—*American War Information Service.*

THE SCHOOL DOCTOR AND THE OTOSCOPE

For an experimental period of one year, Dr. Wilson (*Medical Officer,* 1942:68:37), who is Assistant School Medical Officer to the City of Leicester, used the electric otoscope as a routine measure in the examination of school children in the City of Leicester. The results of these otoscopic examinations were as follows:—

Number of ears examined 4018 (in 2009 children), number of defective ears 729, number of drums obscured by wax 362, number of drums obscured by otitis externa 13, and total number of drums seen and found to be abnormal 354.

Analysis of defects: Drums showing evidence of past otitis:—Retraction 27, scarring 104, dry perforations 129, dullness 7, "chalk patch" 6 and old mastoid scar 2. Drums showing evidence of active middle ear disease:—congestion 58, catarrhal exudate 4, discharging mastoid 1 and perforations with otorrhœa 16 (of which nine had no external discharge).

Children showing abnormal drums due to active disease should be sent for treatment. The most important condition from the point of view of the School Medical Officer is otorrhœa, especially in children who have no external discharge and would, therefore, not have been diagnosed unless an otoscope had been used. During the experimental period of 1 year the author sent 67 cases for treatment. This represented 3·3% of the total number of children seen. Inquiry revealed that other medical officers in the same city who did not employ the otoscope as a routine measure referred only 1·7% of children for treatment of ear disease.

The author concludes that the examination of ears is as valuable as any other part of medical inspection. It is most important that any abnormality should be diagnosed at the earliest possible moment to prevent deafness and recurring disability in children. It is hoped that the use of the otoscope will very soon be universal.—*British Medical Information Service.*

SYNTHESIS OF SANTONIN

Paranjape, Phalnikar, Nargund and Bhidi of the Maharaja Pratapsingh Chemical Laboratory, Sir Parashurambhan College, Poona, write that although the chemical constitution of santonin has been known for a number of years its formal synthesis was not accomplished. In 1930 Clemo and Haworth (J. C. S. 1930:1110.) synthesised desmotroposantonin by a long series of reactions. The synthesis of santonin has been now accomplished in this laboratory by the following reactions:—

5-Chloro-3-keto-Δ^4-tetrahydrobenzene was condensed with diethyl-mono-methyl-malonate which on subsequent hydrolysis and lactonization gave the lactone of 1 hydroxymethyl-2-keto-cyclohexane-acetic acid. This lactone on further formylation, methylation and condensation with ethyl methyl ketone gave racemic santonin which has been identified by its analysis, derivatives and conversion into desmotroposantonin.

DRIED GRASS AS A FOOD SUBSTITUTE

The problem whether grass can be used as a probable substitute for food has been adequately dealt with in Nutritional Observatory, publication of the Heniz Nutritional Research Division of the Mellon Institute, Pittsburgh, Pennslyvania, according to a note in Scientific American. It has been found that the requirements for carotene (provitamin A), ascorbic acid (vitamin C) and iron can be readily met by taking moderate quantities of dried grass. The use of

grass as food (between 4 to 6 ounces) is also recommended in the case of calcium and the vitamin B complex factors, as thiamine, riboflavin and niacin. Dehydrated grass should, however, be used under all circumstances. The flavour, fibre and high water-content and the possibility of eating noxious weeds are the chief reasons against the use of fresh grass. Moreover, human beings, unlike the animals living on grass, are not provided with intestinal bacteria to help digestion.

Dehydration does not destroy any of the nutritive properties of grass. Fresh or dehydrated, grass acts as a laxative and as such the digestive system should be gradually prepared for its consumption. The use of dried grass in small amounts and in a state of fine powder either as an added ingredient in common foods such as bread, or as a supplement to the diet in the form of tablets appears to be the safest recommendation. —*Science and Culture.*

BENGAL RELIEF COMMITTEE

The following is a Statement of Receipts and Payments Account of the Bengal Medical Relief Committee for the period ending 31st May, 1943:—

RECEIPTS	Rs.	As.	P.	PAYMENTS	Rs.	As.	P.
To Donations ..	5,299	4	0	By General Charges a/c (including tram & cooly hire)	34	13	0
„ Suspense Deposit ..	10	0	0	„ Postage ..	36	11	0
				„ Stationery a/c	34	7	0
				„ Medicines ..	25	14	0
				„ Conveyance of Volunteers ..	1,174	15	6
				„ Refreshment for Volunteers ..	10	14	6
				„ Purchase of Equipment for Volunteers ..	118	6	0
				„ Establishment a/c	58	0	0
				„ Telephone a/c	19	13	0
				„ Bank Charges	3	4	0
				„ Printing a/c ..	32	1	0
	5,309	4	0		1,549	3	6
„ Opening Balance* ..	86	14	9	„ Closing Balance	3,846	15	3
	5,396	2	9		5,396	2	9

*Amount brought forward from the last Medical Flood Relief Committee account.

Donations of medicines received (with approximate value) from:—

(1) Corporation of Calcutta, Rs. 75. (2) Messrs. H. Mukherji & Co., Calcutta, Rs. 30. (3) Messrs. Albert David, Ld., Calcutta, Rs. 25. (4) Messrs. Union Drug Co., Ld., Calcutta, Rs. 25. (5) Calcutta Clinical Research Association Ld., Rs. 325. (6) Messrs. Indian Health Institute & Laboratory, Ld., Calcutta, Rs. 254-9-0. (7) Messrs. Bengal Immunity Co., Ld., Calcutta, Rs. 100. (8) Messrs. Standard Pharmaceutical Works, Ld., Calcutta, Rs. 123. (9) The Director of Public Health, Bengal, Rs. 2,700. Total Rs. 3,657-9-0.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors—EDITOR

NEED OF A 'DRINK MORE MILK' CAMPAIGN IN THE COUNTRY AT THE PRESENT TIME

The Editor, *Journal of the I. M. A.*, Calcutta.

Sir,—The nutritional problems of India are too well known. It would be idle to conceal that even in times of peace, balanced diet is not available for the common mass of people. In large areas of the country threre are 'two rice bowls to every three mouths'. Even with those who could afford to eat, the chief defects consist in the 'excessive bulkiness and low nutritive value of the diets'. 'What food is available is nutritionally deficient on account of unscientific method of agriculture, adulteration, or contamination. The result has been that deficiency diseases are common in the country. Sir John Megaw thinks that 'a diet deficient in quality more than in quantity has been the chief cause of the poor physique and proneness to disease of most Indians.'

These defects are easy to discover, but difficult to rectify. The main problems could be grouped under the headings of economics, ignorance, and incompetence.

Now, a medical association is not an economical society. If the disease is there, it has to be treated; the medical body can only take the care of prescribing the cheapest and the best cure in case of the poor. On other aspects of the problem, *viz.*, ignorance and incompetence, the association should undoubtedly lead the way.

Milk is a necessity both in health and disease. In Soviet Russia "compulsory milk-kitchens" are equipments of every child-welfare centre. The provision in England alone, leaving aside Scotland, for 1940 for encouraging 'quality milk production' and its supply to nursing and expectant mothers, infants and school children free or at reduced prices was worth about one and half crores of rupees. At the same time a similar scheme was in vogue in Scotland. The intention of the scheme was that one pint of milk a day free or at a price not exceeding 2d. a pint should be supplied. It will be noted that this was all done after Britain had entered into the war. The food ration of the R.A.F. today is 1 lb. of milk more than what it had been before.

If there can be any solution to India's nutritional problems, the cheapest can be 'Drink more Milk'. In this country, where large sections of people are prejudiced against non-vegetarian diet 'Drink more Milk' is bound to receive the most favourable reception. The nation being mainly agricultural, most of the difficulties that are met in industrialised lands will not be encountered here.

All dangers to infection can be adequately eliminated by heating the milk to boiling point in covered vessels, and by drinking it while hot, served untouched by hand. Boiling does destroy some of the vitamins, particularly heat-labile vitamin C, but almost equally does pasteurisation, condensation, or drying.

The position of the much boosted 'Pasteurisation' *vis-a-vis* 'Drink more Milk' can be conveniently considered here. The theoretical value of the process cannot be disputed. Its actual position, however, as it existed in England three years back can

better be judged from the words of Dr. MacDonald. He says, "Pasteurisation in effect means that Monday's milk is delivered on Thursday". After describing the details of the process, he continues, 'How many milk-producers carry out this process? I may safely say none..... The handling of milk in this country is nothing short of scandalous. With my own eyes I have seen milk, bottled in the street, the milkman producing card board caps from his dirty pockets and sticking them on the bottles. Milk bottles, too, are used by the populace for all sorts of purposes, even urinals. I have also seen milk-cans washed in shallow pond depending for its supply on surface drainage from regularly manured fields." This then was the situation—and probably still is—in the cosmopolitan country of the world. It speaks of the human defects only undoubtedly, but these defects have to be reckoned with in the actual execution of any plan. Thus in actual practice, pasteurisation is likely to prove more dangerous from the false sense of security that it is bound to convey. The British Parliament discussing the subject on July 7, 1939, in the second reading of their Milk Bill rejected the idea that the Government at the moment were under an 'obligation to a national bill for pasteurisation' as 'compulsory pasteurisation was still a controversial matter' and as 'it was not everybody's opinion that pasteurisation provisions were necessary or desirable! Pasteur will turn in his grave if the bare idea is even thought of in this country on any mass scale—at least for a century or two to come. To avert her disaster, it will be far better for India to adapt her old crude ways on the ideas of modern science as best as they can be done than to starve for centuries to come for the achievement of a problematic and elusive ideal. After all, the populace was suffering more from not taking the milk than by taking it in its present way.

The main question, which I had, however, set out to settle was if a need of a 'Drink More Milk' campaign existed in the country today. It will undoubtedly appear that it existed *hoc tempore* more urgently as never before. The war clouds thundering on the horizon should remind the country of the words of Dr. Margaret Balfour. She says, "The Englishman has the advantage that while he can usually fall back on bread, cheese and potatoes when the price of meat rises, the Indian can only reduce his actual amount of food when the cost of his staple food rises." This is less realised in the country than what is its due today. Undoubtedly, 'Grow more Food' will be the first line of defence of the Indian Food. But, is not a second line of defence—defence of the quality—indicated? Whatever may be the actual reasons of the rise in the present prices, the net result has been that every morsel has become dearer and will sooner than later get scarcer if the first line of defence—defence of quantity—may happen to snap—either from under production or from inflation of the market. The quantity can, however, be allowed to suffer provided the quality be proportionately maintained. Dr. Kay says "Given a sufficiency of calories, roughage, and salts in the diet, a pint of milk a day in addition would in practice ensure effective nutrition in almost any human being. In times of emergency, when the diet was much more likely to be restricted than in times of peace, this fact was doubly important." Thus, it will appear that defence of quality—*i.e.*, of milk—will be an unavoidable equipment for the strategy of total defence. The strength of this total defence will not have to be built for the duration of the war only. The peace following in its footsteps will demand no weaker structures.

What, then, is the position in the country today of defence of milk? It will be unfortunate shortsight not to realise from now, when the bombs have not yet started raining, that in times of war in any country it will not be always possible to maintain a steady supply of liquid milk from the udder of the cow or from the kitchen of the home. Under such circumstances, preserved milk—condensed or roller- or spray-dried—will have to be depended upon to meet the need. Methods of conserving milk had greatly improved during the last two decades. Nutritive changes produced in milk by modern methods of drying were relatively small. When stored in an inert gas, dried milk preserved its flavour for 18 months. Dr. Kay says, "In any food defence policy the extensive storage of preserved milk .....commended itself as the wisest complementary step to the storage of wheat or other mainly carbohydrate foods." The conservation of milk in India, can be said to be quite inadequate at the moment.

The position of liquid milk is also not more encouraging. There is adequate production of cereals (showing surplus of 19·5%), and pulses (showing surplus of 2%) to meet the needs of the populace, but the supply of milk is altogether inadequate (showing a deficit of 37%).

The consequent question which thus naturally arises will be—Can milk, with such shortage be depended upon to form the second line of defence? In seeking an answer, one will automatically be led to enquire—If it cannot be milk, what can it be? The medical profession will not probably be able to find the name of any substitute. The situation is, therefore, very disappointing, but not altogether disquieting. The observations of Sir Y. Singh, the present member in charge of Health in the Viceroy's Executive Council, give the only ray of hope. Sir Singh says,. "There is undoubtedly an urgent need to increase our milk supply. It is, however, possible to increase the present livestock and almost double the milk supply by following modern methods of animal husbandry."

A campaign will, therefore, be needed to bear the torch of this message. It may be belated, but there will be no other cause to regret it. It will in no way lead to deplete the available stock—the sense of danger is entirely false as more than the adequate stock already exists in the country in potential form. The campaign, on the other hand, will help to make the people as well as the Government more milk-minded. Only such a mind can lead to bring out the potential stock from its present concealed position to build the necessary second line of defence of the Indian Food and as its corollary, to ensure adequate fodder supply, and last but not the least, to adoption of such measures either to preserve the present arrangements (by which the urban areas get their supply) with a view to prevent any interruption of this essential supply in times of emergency, or to provide for its alternative by conservation of milk. How far such a campaign will aid in the long run to better the economic fabric and help to remove the ignorance of the populace about the value of food, will no doubt, be favourably surmised.

I am etc.

Sadar Hospital,
Muzaffarpore.

Rajendra Prasad, M.B.B.S.,

OBITUARY

D. R. RANJIT SINGH

Major D. R. Ranjit Singh died of heart failure at Allahabad on 8-4-43. He was an active member of the Allahabad Branch of the Indian Medical Association, being the President of the U.P. Provincial Branch in 1935-36.

Capt. S. K. Chaudhuri of the Benares Medical Association (Benares Branch, I.M.A.) who was very intimate with the deceased gives an appreciative account of Major Ranjit Singh in the following lines:

Major Ranjit Singh is no more with us. The gap caused in the profession will be hard to fill. He was a product of the fine old Punjab School which, prior to the establishment of the Medical College at Lucknow, supplied the needs of the U.P., C.P., N.W.F.P. and the Punjab proper. He survived most of his contemporaries who studied with him at the Medical College, Lahore, and succeeded in winning an outstanding position for himself. He made many friends, thanks to his interest in spheres of life far beyond the limits of his profession. His services as a citizen were rewarded by his being made President of the Allahabad Improvement Trust. He enjoyed the confidence of the public and Government alike. He represented the independent medical profession on the U.P. Medical Council and other institutions of importance. He was an exponent of Indian music; his efforts to promote this art will long be remembered by lovers of music in these provinces. His zeal for the spread of education was well known. The Kayasth Pathshala is indebted to him for the position it occupies. His wide sympathy, sociable habits, charming manners and sense of humour and fair play easily put him far above those among whom he lived. His services in the last war might have been more suitably recognised. He was a soldier and one recalls the line "old soldiers never die but simply fade away".

R. N. BHATIA

Rai Bahadur Dr. R. N. Bhatia, F.R.C.S., died at his residence in Lucknow on 17-2-43. He was a member of the Indian Medical Association since 22-2-33. A well-known and popular surgeon of the Province, he was for sometime Professor of Surgery, King George's Medical College, Lucknow. His death is a great loss to the Association, the profession and the public.

P. L. WHIG

Dr. P. L. Whig, M.B., M.R.C.P., D.P.H., D.T.M, & S., T.D,D., died of heart failure on 21-4-43. He was an active member of the Cawnpur Branch of the Indian Medical Association since 25-2-41.

MUKHERJEE—THE ROLE OF THE OBSTETRICIAN AND GYNÆCOLOGIST IN FUTURE INDIA

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considerable advance in such work. In our country it is a well-known fact that most gynæcological complaints are due to bad midwifery. We have through these obstetric societies to devise plans of giving effective antenatal and postnatal care to our women. It is no doubt a stupendous task but it is in this way alone that we can prevent much maternal suffering and mortality. It is a shame that in India almost 99 per cent of expectant mothers go to labour without any antenatal care at all. A beginning at a large scale maternity and child-welfare work has to be made and who are better qualified to do such work than we ourselves? Let us make a beginning through these obstetric societies.

At every step, however, we are conscious that if we are to discharge our duty to the society we must be helped by the State. The necessity of State-aid in our task of building up a happy nation through the rearing of a healthy motherhood in the country can hardly be over-emphasised. Maternity and child-welfare work in India has not yet received at the hands of the Government the attention it deserves. Bare doles hitherto meted out are not enough. Vast sums of money are needed for this most important work if the nation is to thrive and look forward to a happy future. The medical science in almost all its departments has made rapid progress. Particularly in the domain of biochemistry, physical therapy and preventive medicine, the progress has been considerable. It is up to any civilised State to see that the fruits of all such advances in science are made available to the community. It is very unfortunate indeed that even to-day the importance of the preventive aspect of maternity is not realised in full either by the State or the public. Even the general physician is often forgetful of it. I cannot say either that the Public Health authorities have been as much alive as they should be to this aspect. It is high time that we thought much more seriously of the welfare of the helpless Indian mothers and prospective mothers in the rural areas—for they are the back-bone of the nation. The nation in India lives in the millions of villages. No work of national regeneration in the true sense of the term is possible if we forget or ignore them.

Ladies and Gentlemen, I do not want to detain you any more. I shall conclude with one last word. We must remember that in any future work of social and national reconstruction we, the obstetricians and gynæcologists, have a definite and vital role to perform. We are the natural custodians of the health and well-being of millions of our women and children. Our mission is sacred and our responsibility great. Let us, in whatever we do, bear these thoughts in mind. Let us jointly endeavour to achieve our common ideal—the health and happiness of the women of India.

May Providence guide us in our task!

"या देवी सर्व्वभूतेषु मातृरुपेण संस्थिता।
नमस्तस्य नमस्तस्यै नमस्तस्यै नमो नमः॥"